EXHIBIT C17

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RULE 26 EXPERT REPORT OF DR. GHASSAN M. SAED

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Molecular basis for the association of talcum powder use with increased risk of ovarian cancer.

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Qualifications

In this report, I describe the role of oxidative stress in the pathogenesis and behavior of ovarian cancer, as well as describe the biological effects of talcum powder on normal ovarian and fallopian tube cells, macrophages, and ovarian cancer cells.

I am an Associate Professor with tenure at Wayne State University in Detroit, Michigan, where I am Director of Ovarian Cancer Research. I am a faculty member in the Departments of Obstetrics & Gynecology, Cell Biology, and Anatomy & Physiology at Wayne State School of Medicine. I am also a Member of the Karmanos Cancer Institute, Molecular Biology and Genetics Program.

I received a Ph.D. in Molecular Biology at the University of Essex, Colchester, England in 1986. My postgraduate training included a Fellowship in Immunopathology at the University of Michigan, Ann Arbor from 1992-1993 and a Fellowship in Molecular Biology at the Henry Ford Hospital in Detroit, Michigan from 1988-1990. I joined the faculty at Wayne State School of Medicine in 1998.

My laboratory investigates the role of oxidative stress in the pathogenesis of ovarian cancer. This concentration arose from my original research that focused on the molecular mechanisms involved in the pathogenesis of tissue fibrosis and the need to compare the effects of oxidative stress on a malignant overgrowth versus a benign overgrowth, specifically postoperative adhesions.

My research in ovarian cancer has resulted in the identification of biomarkers for assessing the progression and metastasis of ovarian cancer. The major outcome of my work with fibrosis and postoperative adhesions, in addition to the development of the ex-vivo model for adgestiosn, was the development and characterization of the adhesion phenotype in cell culture. Additionally,

the cell culture system was used to test the hypothesis that hypoxia is the trigger for the development of postoperative adhesions.

I have taught numerous undergraduate, graduate, medical students, residents, and fellows. Many of these have received research awards, published important papers, and accepted prestigious academic faculty positions. I have been the recipient of national and international grants and contracts from organizations including the American Association for Cancer Research (AACR), NIH/NICHD, U.S. Department of Defense, the Ovarian Cancer Research Fund Alliance, the Michigan Ovarian Cancer Alliance, and other ovarian cancer foundations. I have been a prolific publisher and presenter at scientific meetings. I have been an author on 136 original studies published in peer-reviewed journals with additional review articles, and book chapters. Recently, I published a review article in the journal, Gynecologic Oncology titled, "Updates of the role of oxidative stress in the pathogenesis of ovarian cancer" and a textbook chapter titled "New insights into the Pathogenesis of Ovarian Cancer: Oxidative Stress" summarizing my research in this area. My CV is attached as Exhibit A. In addition to the references included in this report, the materials I reviewed are attached as Exhibit B. My fees and prior testimony are attached as Exhibit C.

Ovarian cancer

Ovarian cancer is the most lethal gynecologic malignancy and ranks fifth in cancer deaths among women diagnosed with cancer[1]. Epithelial ovarian cancer (EOC) has long been considered a heterogeneous disease with respect to histopathology, molecular biology, and clinical outcome[1,2]. It comprises at least five distinct histological subtypes, the most common and well-studied being high-grade serous ovarian cancer. In the last decade, researchers have proposed the theory that many ovarian cancers arise from the distal fallopian tubes. For this reason, as well as the similarities in pathogenesis, presentation, treatment, and prognosis, fallopian tube, ovarian, and

peritoneal cancer are generally treated as a single entity. Although surgical techniques and treatments have advanced over the years, the prognosis of EOC remains poor, with a 5-year survival rate of 50% in advanced stage [2]. This is largely due to the lack of early warning symptoms and screening methods, and the development of chemoresistance [1,2]. Ovarian cancer is known to be associated with germline mutations in the BRCA1 or BRCA2 genes, but with a rate of only 20-40%, suggesting the presence of other unknown mutations in other predisposition genes [3]. Additional genetic variations including single nucleotide polymorphisms (SNPs) have been described to act as low to moderate penetrant alleles that contribute to ovarian cancer risk [3,4]. Non-synonymous SNPs substitute encoded amino acids in proteins and are more likely to alter the structure, function, and interaction of the protein [4]. The pathophysiology of EOC is not fully understood but has been strongly associated with inflammation and the resultant oxidative stress[5].

Oxidative stress

Homeostasis, the balance between the production and elimination of oxidants, is maintained by mechanisms involving oxidants and antioxidant enzymes and molecules. If this balance is altered, it leads to an enhanced state of oxidative stress that alters key biomolecules and cells of living organism [5]. Oxidant molecules are divided into two main groups; oxygen-derived or nitrogen-containing molecules. Oxygen-derived molecules, also known as reactive oxygen species (ROS), include free radicals such as hydroxyl ($HO^{\bullet}$), superoxide ($O_2^{\bullet -}$), peroxyl ($RO_2^{\bullet}$), and alkoxyl ($RO^{\bullet}$), as well as oxidizing agents such as hydrogen peroxide (H_2O_2), hypochlorous acid (HOCl), ozone (O_3), and singlet oxygen ($^1O_2$) that can be converted to radicals [5,6]. Nitrogen containing oxidants, also known as reactive nitrogen species (RNS), are derived from nitric oxide (NO) that is produced in the mitochondria in response to hypoxia [5]. Exposure to inflammation, infection, carcinogens, and toxicants are major sources of ROS and RNS, *in vivo* [5-8]. Additionally,

RNS and ROS can be produced by various enzymes including cytochrome P450, lipoxygenase, cyclooxygenase, nicotinamide adenine dinucleotide phosphate (NAD(P)H) oxidase complex, xanthine oxidase (XO), and peroxisomes (Figure 1) [5,7,9].

To maintain the redox balance, ROS and RNS are neutralized by various important enzyme systems including superoxide dismutase (SOD), catalase (CAT), glutathione S-transferase (GST), glutathione (GSH), thioredoxin coupled with thioredoxin reductase, glutaredoxin, glutathione peroxidase (GPX), and glutathione reductase (GSR) [6]. Superoxide dismutase is known to convert $O_2^{\bullet -}$ to H_2O_2, which is then converted to water by CAT. Glutathione S-transferase is involved in detoxification of carcinogens and xenobiotics by catalyzing their conjugation to GSH that will aid in expulsion from the cell [6]. Indeed, the GSH-to-oxidized-GSH (GSH/GSSG) ratio is a good indicator of cellular redox buffering capacity [10,11]. Under enhanced oxidative stress, the GSH/GSSG complex is known to stimulate the activity of the GS-X-MRP1 efflux pump, which removes toxins from cells. This mechanism has been investigated in the development of resistance to chemotherapeutic drugs [10,11].

Ovarian Cancer Cells Manifest a Persistent Pro-Oxidant State

Recent evidence demonstrates that oxidative stress is a critical factor in the initiation and development of several cancers, including ovarian cancer [12,13]. Consistently, it has been reported that ovarian cancer patients manifest significantly decreased levels of antioxidants and higher levels of oxidants [12-17]. An enhanced redox state, resulting from increased expression of key pro-oxidant enzymes and decreased expression of antioxidant enzymes, has been extensively described in epithelial ovarian cancer (EOC) [16-18]. My laboratory has previously reported that MPO, a hemoprotein present solely in myeloid cells that acts as a powerful oxidant, and iNOS, a key pro-oxidant enzyme, are highly expressed and co-localized to the same cell in EOC cells [17]. These two

enzymes, MPO and iNOS, work together to inhibit apoptosis, a hallmark of ovarian cancer cells. Apoptosis, or programmed cell death, refers to the normal and controlled death of cells. Myeloperoxidase acts as powerful oxidant enzyme in EOC cells through the utilization of nitric oxide (NO) produced by iNOS as a one-electron substrate generating NO^+, a labile nitrosylating species[19-21]. My laboratory was the first to report that MPO was expressed by EOC cells and tissues[17]. Silencing MPO gene expression utilizing MPO specific siRNA induced apoptosis in EOC cells through a mechanism that involved the S-nitrosylation of caspase-3 by MPO[17]. Additionally, there is compelling evidence that MPO serves as a source of free iron under oxidative stress, where both NO^+ and superoxide are elevated[19]. Iron reacts with hydrogen peroxide (H_2O_2) and catalyzes the generation of highly reactive hydroxy radical ($HO^\bullet$), thereby increasing oxidative stress, which in turn increases free iron concentrations by the Fenton and Haber–Weiss reaction[19,21]. Additionally, my laboratory has highlighted the potential benefits of the combination of serum MPO and free iron as biomarkers for early detection and prognosis of ovarian cancer[14]. EOC cells are also characterized by enhanced expression of NAD(P)H oxidase, a potent oxidant enzyme that is known to be the major source of $O_2^{\bullet-}$ in the cell. Such high levels of $O_2^{\bullet-}$ combined with significantly high levels of NO generates peroxynitrite, another powerful nitrosylation and nitration agent, which modifies proteins and DNA structure and function in cells [22].

A reliable screening and detection method based on molecular profiles for ovarian cancer has not yet been developed because the disease exhibits a wide range of morphological, clinical and genetic variations during its progression. The search for non-invasive, cost-effective ovarian cancer biomarker tests has been ongoing for many years. Immunizations of mice with ovarian cancer cells has led to hybridoma validation by ELISA, while flow cytometry analysis permitted the discovery of cancer antigen (CA)-125 (the only marker currently used in clinical practice) and

mesothelin [23]. Furthermore, the screening of an array of 21,500 unknown ovarian cDNAs hybridized with labeled first-strand cDNA from ten ovarian tumors and six normal tissues led to the discovery of human epididymis protein 4 (HE4) [24]. Most interestingly, HE4 is overexpressed in 93% of serous and 100% of endometrioid EOCs, and in 50% of clear cell carcinomas, but not in mucinous ovarian carcinomas [25]. Thus, HE4 was identified as one of the most useful biomarkers for ovarian cancer, although it lacked tissue-specificity [24,26-28]. Secreted HE4 high levels were also detected in the serum of ovarian cancer patients [29]. Additionally, combining CA-125 and HE4 is a more accurate predictor of malignancy than either alone [30-32]. The discovery of MPO expression in ovarian EOC cells and tissues was surprising, as it is only expressed by cells of myeloid origin. Intriguingly, my laboratory has previously reported that the combination of serum MPO and free iron may serve as biomarkers for early detection of ovarian cancer [14].

Common Polymorphisms in Redox Enzymes are Associated with Ovarian Cancer.

A single nucleotide polymorphism (SNP) occurs as a result of gene point mutations with an estimated frequency of at least one in every 1000 base pairs that are selectively maintained and distributed in populations throughout the human genome [33]. An association between common SNPs in oxidative DNA repair genes and redox genes with human cancer susceptibility has been established [7]. Common SNPs in the redox enzymes are known to be strongly associated with an altered enzymatic activity in these enzymes, and helps explain the enhanced redox state that has been linked to several malignancies, including ovarian cancer [12,16]. Additionally, it helps explain the observation of significantly decreased apoptosis and increased survival of EOC cells [17]. It is therefore critical to determine the exact effect of common SNPs in various redox enzymes on all process involved in the development of the oncogenic phenotype [34-37]. Such studies can be linked to other studies focusing on determining the effects of genes involved in carcinogen metabolism

(detoxification and/or activation), redox enzymes, and DNA repair pathways[36]. Numerous SNPs associated with change of function have been identified in antioxidant enzymes including *CAT*, *GPX1*, *GSR*, and *SOD2* [35,37]. Additionally, the association between genetic polymorphisms in genes with anti-tumor activity and those involved in the cell cycle has been reported in ovarian cancer [38,39]. Recently, several genetic variations have been identified in genome-wide association studies (GWAS), and were found to act as low to moderate penetrant alleles, which contribute to ovarian cancer risk, as well as other diseases [4,40].

There is now an association of specific SNPs in key oxidant and anti-oxidant enzymes that impact increased risk of ovarian cancer and/or overall survival of patients with ovarian cancer [34,35]. A common SNP that reduced CAT activity (rs1001179) was utilized as a significant predictor of death when present in ovarian cancer patients and was also associated with increased risk for breast cancer [34,35,37,41]. This SNP is also linked to increased risk, survival, and response to adjuvant treatment of cancer patients, including ovarian [34,42]. Another common SNP that reduced CYBA activity (rs4673) was also reported to be associated with an increased risk for ovarian cancer [34,35]. The mutant genotype of the *CYBA* gene has been shown to both decrease and increase activity of the protein, thereby altering the generation of $O_2^{\bullet-}$ [34,35]. Moreover, functionally distinct *MPO* polymorphisms, such as (rs2333227) have been linked to relative increased risk for development of ovarian cancer as well as other cancers [34,35,43]. Additional SNPs that influenced the risk of EOC have been successfully identified from the GWAS studies including rs3814113 (located at 9p22, near BNC2), rs2072590 (located at 2q31, which contains a family of HOX genes), rs2665390 (located at 3q25, intronic to TIPARP), rs10088218 (located at 8q24, 700 kb downstream of MYC), rs8170 (located at 19p13, near MERIT40), and rs9303542 (located at 17q21, intronic to SKAP1)

[34,35]. Thus, the genetic component of increased ovarian cancer risk may be attributed to SNPs that result in point mutations in the redox genes and potentially other genes [44].

Chemoresistance is Associated with Point Mutations in Key Redox Enzymes in EOC cells

To date, the acquisition of chemoresistance in ovarian cancer is being investigated. The enhanced oxidant state reported in chemoresistant EOC cells is likely linked to point mutations in key redox enzymes [35]. Chemoresistant EOC cells manifested increased levels of CAT, GPX, and iNOS and decreased levels of GSR, SOD, and NAD(P)H oxidase as compared to their sensitive counterparts [35]. Interestingly, chemoresistant EOC cells, and not their sensitive counterparts, manifested specific point mutations that corresponded to known functional SNPs, in key redox enzymes including *SOD2* (rs4880), *NOS2* (rs2297518), and *CYBA* (rs4673) [45]. However, altered enzymatic activity for CAT and GSR observed in chemoresistant EOC cells did not correspond to the specific SNP of interest in those enzymes, indicating involvement of other possible functional SNPs for those enzymes [35]. Coincidently, chemotherapy treatment induced point mutations that happen to correspond to known functional SNPs in key oxidant enzymes subsequently led to the acquisition of chemoresistance by EOC cells. Indeed, the induction of specific point mutations in *SOD2* or *GPX1* in sensitive EOC cells resulted in a decrease in the sensitivity to chemotherapy of these cells [35]. In fact, the addition of *SOD* to sensitive EOC cells during chemotherapy treatment synergistically increased the efficacy to chemotherapy [35].

Alternatively, the observed nucleotide switch in response to chemotherapy in EOC cells may be the result of nucleotide substitution, a process that includes transitions, replacement of one purine by the other or that of one pyrimidine by the other, or transversions, replacement of a purine by a pyrimidine or vice versa [35]. Actually, hydroxyl radicals are known to react with DNA causing the formation of many pyrimidine and purine-derived lesions [35]. The oxidative damage to 8-Oxo-

2'-deoxyguanosine, a major product of DNA oxidation, induces genetic alterations in oncogenes and tumor suppressor genes that have been involved in tumor initiation and progression [35]. A GC to TA transversion has been reported in the *ras* oncogene and the *p53* tumor suppressor gene in several cancers. However, the GC to TA transversion is not unique to hydroxy-2'-deoxyguanosine, as CC to TT substitutions have been identified as signature mutations for oxidants and free radicals [35]. Moreover, the observed nucleotide switch in response to chemotherapy in EOC cells can be due to the fact that acquisition of chemoresistance generates an entirely different population of cells with a distinct genotype. Hence, chemotherapy kills the bulk of the tumor cells leaving a subtype of cancer cells with ability for repair and renewal, known as cancer stem cells (CSCs) [35]. Indeed, cancer stem cells have been isolated from various types of cancer including leukemia, breast, brain, pancreatic, prostate, ovary and colon [35]. Interestingly, CSC populations were present in cultures of SKOV-3 EOC cells and have been shown to be chemoresistant in nature [35].

Talcum powder and increased risk of ovarian cancer

Talcum powder is made from talc, a mineral containing mainly of the elements magnesium, silicon, and oxygen. In its natural form, some talc contains asbestos. Talc and asbestos are both silicate minerals; the carcinogenic effects of asbestos have been extensively studied and documented in the medical literature [46,47]. Asbestos fibers in the lung initiate an inflammatory and scarring process, and it has been proposed that ground talc, as a foreign body, initiates a similar inflammatory response and it has been proposed that ground talc, as a foreign body, might initiate an inflammatory response [48,47]. There has been concern about a possible link between talcum powder usage in the genital and ovarian cancer, as well as lung cancer in workers exposed to talc in an occupational setting [49]. Studies that exposed lab animals (rats, mice, and hamsters) to asbestos-free talcum powder in various ways have had mixed results, with some showing tumor

formation and others finding only inflammation [50,51]. The International Agency for Research on Cancer (IARC) is part of the World Health Organization (WHO). Its major goal is to identify causes of cancer. Based on limited evidence from human studies of a link to ovarian cancer, IARC classified the perineal (genital) use of talc-based body powder (not containing asbestiform fibers) as "possibly carcinogenic to humans." (Group 2b) [88]. Talcum powder containing asbestos and fibrous talc are both considered carcinogenic (Group 1) by IARC [89].

The association between perineal talc powder dusting and ovarian cancer has been studied in at least 25 case-control studies, three cohort studies, six meta-analyses and one pooled study [73]. Although the cohort studies individually did not show a statistically significant increased risk of ovarian cancer with talcum powder usage, the case-control studies overall and the meta-analyses show a consistent and significant increased risk. This risk is estimated to be 30-40%. The studies have shown conflicting results regarding the presence of a dose-response, largely due to the failure of many studies to obtain necessary information on the frequency and duration of usage and the inherent challenge of quantifying actual exposure. Although migration/transport of particles through the genital tract is universally accepted and the inflammatory nature of talcum powder consistently demonstrated, the exact mechanism for carcinogenesis had not been conclusively elucidated. For these reasons, there has been some reluctance in the scientific community to accept talcum powder as a causative risk factor for the development of ovarian cancer. The most recent meta-analysis, reported by Penninkilampi and Eslick in 2017, found that any perineal talc use was associated with a statistically significant increased risk of ovarian cancer (OR = 1.31). More than 3600 lifetime applications (OR = 1.42) were slightly more associated with ovarian cancer than <3600 (OR = 1.32). An association with ever use of talc was found in case–control studies (OR = 1.35), but not cohort studies (OR = 1.06). However, cohort studies found an association between

talc use and invasive serous type ovarian cancer (OR = 1.25), the most common and most lethal subtype. In the opinion of the authors, meta-analysis is the highest level of evidence in this setting because of the need for a large number of cases and long-term follow-up in a relatively rare form of cancer with a lengthy latency period. The authors of this meta-analysis suggested that cellular injury, oxidative stress, and local increase in inflammatory mediators might be the mechanism by which talcum powder promotes carcinogensis, but that this mechanism was unclear. They recognized the "substantial need for further research on a potential mechanism by which ovarian cancer may be caused by talc, as this will allow a causal relationship to be established or rejected with more certainty" [73].

In addition to epidemiological studies, the claim that regular use of talcum powder for perineal hygiene purpose is associated with an increased risk of ovarian cancer is based on several reports confirming the presence of talc particles in the ovaries and other parts of the female reproductive tract as well as in lymphatic vessels and tissues of the pelvis [45,75]. Henderson first reported the presence of talc particles in ovaries in 1971. A study by Cramer, et al has reported the presence of talc in pelvic lymph nodes of a woman with ovarian cancer who used talc daily for 30 years[45]. The ability of talc particles to migrate through the genital tract to the distal fallopian tube and ovaries is well accepted [45,75].

It has been suggested that the associations between perineal talc dusting and ovarian cancer might be explained by the induction of ANTI-MUC1 antibodies [57]. Additionally, in a previous study by Shukla et.al., whereby human mesothelial cells (LP9/TERT-1) were exposed to low and high (15 and 75 mm2/cm2 dish) equal surface area concentrations of nonfibrous talc for 8 or 24 hours, the authors found that nonfibrous talc at low concentrations to cause an increase in the

expression of Activating Transcription Factor 3 (ATF3) at 8 hours and no changes at 24 hours, whereas expression levels of 30 genes were elevated at 8 hours at high talc concentrations [78].

My laboratory undertook research to determine whether or not there was a molecular basis for the observed association between talcum powder and ovarian cancer. If a biological effect was demonstrated, we hoped to define the mechanism in detail. Issues like this one, relating to the pathogenesis of ovarian cancer and the relationship between inflammation and other pathological conditions in the female reproductive system as well as cancer, have been the focus of my laboratory for many years.

Findings from recent research from our laboratory relating to the effects of talcum powder exposure *in vitro*

The following is a description of the methodology used:

Cell Lines: Ovarian cancer cells: SKOV-3 (ATCC), A2780 (Sigma Aldrich), and TOV112D (a kind gift from Gen Sheng Wu at Wayne State University, Detroit, MI) [25]. Normal cell lines: human macrophage cells (EL-1, ATCC), human primary normal ovarian epithelial cells (Cell Biologics), Human Ovarian Epithelial Cells (HOSEpiC, ScienCell Research Laboratories, Inc.) immortalized human fallopian tube secretory epithelial cells (FT33-shp53-R24C, Applied Biological Materials). All cells were grown in media and conditions following manufacturer's protocol. EL-1 cells were grown in IMDM media (ATCC) supplemented with 0.1 mM hypoxanthine and 0.1 mM thymidine solution (H-T, ATCC) and 0.05mM β-mercaptoethanol. SKOV-3 EOC cells were grown in HyClone McCoy's 5A medium (Fisher Scientific), A2780 EOC cells were grown in HyClone RPMI-1640 (Fisher Scientific), and both TOV112D EOC cells were grown in MCDB105 (Cell Applications) and Medium 199 (Fisher Scientific) (1:1). All media was supplemented with fetal bovine serum (FBS, Innovative Research) and penicillin/streptomycin

(Fisher Scientific), per their manufacturer specifications. Human primary normal ovarian epithelial cells were grown in Complete Human Epithelial Cell Medium (Cell Biologics).

Treatment of cells: Talcum powder (Fisher Scientific, Catalog #T4-500, Lot#166820) or Johnson's Baby Powder (Johnson & Johnson, #30027477, Lot#13717RA) was dissolved in DMSO (Sigma Aldrich) at a concentration of 500 mg in 10 ml and was filtered with a 0.2 µm syringe filter (Corning). Sterile DMSO was used as a control for all treatments. Cells were seeded in 100 mm cell culture dishes (3×10^6) and were treated 24 hours later with 0, 5, 20, or 100 µg/ml of talc for 48 hours. Cell pellets were collected for RNA, DNA, and protein extraction. Cell culture media was collected for CA-125 analysis by ELISA.

Real time RT-PCR: Total RNA was extracted from all cells using the RNeasy Mini Kit (Qiagen) according to the protocol provided by the manufacturer. Measurement of the amount of RNA in each sample was performed using a Nanodrop Spectrophotometer (Thermo Fisher Scientific). A 20 µL cDNA reaction volume containing 0.5 µg RNA was prepared using the SuperScript VILO Master Mix Kit (Life Technologies), as described by the manufacturer's protocol. Optimal oligonucleotide primer pairs were selected for each target using Beacon Designer (Premier Biosoft, Inc., Table 1). Quantitative RT-PCR was performed using the EXPRESS SYBR Green ER qPCR Supermix Kit (Life Technologies) and the Cepheid 1.2f Detection System as previously described[24]. Standards with known concentrations and lengths were designed specifically for β-actin (79 bp), CAT (105 bp), iNOS (89 bp), GSR (103 bp), GPX1 (100 bp), MPO (79 bp), and SOD3 (84 bp), allowing for construction of a standard curve using a 10-fold dilution series[26]. A specific standard for each gene allows for absolute quantification of the gene in number of copies, which can then be expressed per microgram of RNA. All samples

were normalized to the housekeeping gene, β-actin. A final melting curve analysis was performed to demonstrate specificity of the PCR product.

Protein Detection: Cell pellets were lysed utilizing cell lysis buffer (20 mM Tris-HCl (pH 7.5), 150 mM NaCl, 1 mM Na2EDTA, 1 mM EGTA, 1% Triton, 2.5 sodium pyrophosphate, 1 mM beta-glycerophosphate, 1 mM Na3VO4, 1 µg/ml leupeptin) containing a cocktail of protease inhibitors. Samples were centrifuged at 13000 rpm for 10 minutes at 4°C. Total protein concentration of cell lysates from control and talc-treated cells was measured with the Pierce BCA Protein Assay Kit (Thermo Scientific, Rockford, Illinois) per the manufacturer's protocol. *Detection of protein/activity by ELISA:* ELISA kits for each target were purchased and used according to the manufacturer's protocol. The following ELISA kits were purchased from Cayman Chemical, Ann Arbor, MI: CAT, SOD3, GSR, GPX1, and MPO. Nitrite ($NO2^-$)/nitrate ($NO3^-$) were determined spectrophotometrically by measuring their absorbance at 210 nm after separation by HPLC with standard $NO2^-$/$NO3^-$ as previously reported[19]. The analysis was performed by a HPLC system (Shimadzu Scientific Instruments, Inc.) including a LC-10ADV pump, frc-10A injector and DGU-14A degasser. Nitrite/nitrate were detected using an RF-10 XL fluorescence detector with 210 nm excitation and 440 nm emission. CA-125 protein levels were measured in cell media by ELISA from Ray Biotech according to the manufacturer's protocol.

TaqMan® SNP Genotyping Assay: DNA was isolated utilizing the EZ1 DNA Tissue Kit (Qiagen Valencia, CA) for EOC cells according the manufacturer's protocols. The TaqMan® SNP Genotyping Assay Set (Applied Biosystems, Carlsbad, CA) (NCBI dbSNP genome build 37, MAF source 1000 genomes) were used to genotype the SNPs described in Table 1. The Applied Genomics Technology Center (AGTC, Wayne State University, Detroit, MI) performed these

assays. Analysis was done utilizing the QuantStudioTM 12 K Flex Real-Time PCR System (Applied Biosystems).

Statistical Analysis: Normality was examined using the Kolmogorov-Smirnov test and by visual inspection of quantile-quantile plots. Because most of the data were not normally distributed, differences in distributions were examined using the Kruskal-Wallis test. Generalized linear models were fit to examine pairwise differences in estimated least squares mean expression values by exposure to 0, 5, 20 or 100 ug/ml of Talc. We used the Tukey-Kramer adjustment for multiple comparisons, and the regression models were fit using log2 transformed analyte expression values after adding a numeric constant '1' to meet model assumptions while avoiding negative transformed values. P-values below 0.05 are statistically significant.

Research Findings: Recent studies from our laboratory have shown conclusively that talcum powder alter key redox and inflammatory markers, enhance cell proliferation, and inhibit apoptosis in EOC cells, which are hallmarks of ovarian cancer. More importantly, this effect is also manifested by talcum powder in normal cells, including surface ovarian epithelium, fallopian tube, and macrophages. Oxidative stress has been implicated in the pathogenesis of ovarian cancer, specifically, by increased expression of several key pro-oxidant enzymes such as iNOS, MPO, and NAD(P)H oxidase in EOC tissues and cells as compared to normal cells indicating an enhanced redox state, as we have recently demonstrated [19]. This redox state is further enhanced in chemoresistant EOC cells as evident by a further increase in iNOS and $NO2^-/NO3^-$ and a decrease in GSR levels, suggesting a shift towards a pro-oxidant state [19]. Antioxidant enzymes, key regulators of cellular redox balance, are differentially expressed in various cancers, including ovarian [19,79]. Specifically, GPX expression is reduced in prostate, bladder, and estrogen receptor negative breast cancer cell lines as well as in cancerous tissues from the kidney. However, GPX

activity is increased in cancerous tissues from breast [79]. Glutathione reductase levels, on the other hand, are elevated in lung cancer, although differentially expressed in breast and kidney cancerous tissues [5,80]. Similarly, CAT was decreased in breast, bladder, and lung cancer while increased in brain cancer [81-83]. Superoxide dismutase is expressed in lung, colorectal, gastric ovarian, and breast cancer, while decreased activity and expression have been reported in colorectal carcinomas and pancreatic cancer cells [83-86]. Collectively, this differential expression of antioxidants demonstrates the unique and complex redox microenvironment in cancer. Glutathione reductase is a flavoprotein that catalyzes the NADPH-dependent reduction of oxidized glutathione (GSSG) to GSH. This enzyme is essential for the GSH redox cycle which maintains adequate levels of reduced cellular GSH. A high GSH/GSSG ratio is essential for protection against oxidative stress. Treatment with talc significantly reduced GSR in normal and cancer cells, altering the redox balance. Likewise, GPX is an enzyme that detoxifies reactive electrophilic intermediates and thus plays an important role in protecting cells from cytotoxic and carcinogenic agents. Overexpression of GPX is triggered by exogenous chemical agents and reactive oxygen species, and is thus thought to represent an adaptive response to stress [80]. Treatment of normal and cancer cells with talc significantly reduced GPX, which compromised the overall cell response to stress.

We have previously reported that EOC cells manifest increased cell proliferations and decreased apoptosis [19]. Consistent with these findings, recent studies from my laboratory have shown that talc enhances cell proliferation and induces an inhibition in apoptosis in EOC cells, but more importantly in normal cells, suggesting talc is a stimulus to the development of the oncogenic phenotype. We also previously reported a cross-talk between iNOS and MPO in ovarian cancer which contributed to the lower apoptosis observed in ovarian cancer cells [17,19]. Collectively, we now have substantial evidence demonstrating that altered oxidative stress may play a role in

maintaining the oncogenic phenotype of EOC cells. Treatment of normal or ovarian cancer cells with talc resulted in a significant increase in MPO and iNOS, supporting the role of talc in the enhancement of a pro-oxidant state that is a major cause in the development and maintenance of the oncogenic phenotype.

Furthermore, CA-125, which exists as a membrane-bound and secreted protein in epithelial ovarian cancer cells, has been established as a biomarker for disease progression and response to treatment [2]. CA-125 expression was significantly increased from nearly undetectable levels in controls to values approaching clinical significance (35 U/ml in postmenopausal women[87]) in talc treated cell lines without the physiologic effects on the tumor microenvironment one would expect to be present in the human body, highlighting the implications of the pro-oxidant states caused by talc alone.

To elucidate the mechanism by which talc alters the redox balance to favor a pro-oxidant state not only in ovarian cancer cells, but more importantly in normal cells, my laboratory examined selected known gene mutations in key oxidant and antioxidant enzymes. These mutations correspond to specific SNPs that are known to be associated with altered enzymatic activity and increased cancer risk [34,35]. Results show that the *CAT* SNP (rs769217) which results in decreased enzymatic activity was induced in all normal cell lines tested and in TOV112D EOC lines. However, the *CAT* mutation was not detected in A2780 or SKOV-3 cell lines. Nevertheless, our results confirm a decrease in CAT expression and enzymatic activity in all talc treated cells, indicating the existence of other *CAT* SNPs. However, the *SOD3* (rs2536512) and *GSR* (rs8190955) SNP genotypes were not detected in any cell line, yet SOD3 and GSR activity and expression were decreased in all talc treated cells, again suggesting the presence of other SNPs. Our results have also shown that all cells, except for HOSEpiC cells, manifest the SNP genotype

of *GPX1* (C/T) before talc treatment. Intriguingly, talc treatment reversed this SNP genotype to the normal genotype. Consistent with this finding, it has previously been reported that acquisition of chemoresistance by ovarian cancer cells is associated with a switch from the *GPX1* SNP genotype to the normal *GPX1* genotype [35]. It is not understood why a *GPX1* SNP genotype predominates in untreated normal and ovarian cancer cells. Additionally, our results showed that talc treatment was associated with a genotype switch from common C/C genotype in *NOS2* in untreated cells to T/T, the SNP genotype, in talc treated cells, except in A2780 and TOV112D. Nevertheless, our results confirm an increase in iNOS expression and enzymatic activity in all talc treated cells, again suggesting the existence of other *NOS2* SNPs. Collectively, these findings demonstrate that talc treatment induced gene point mutations that happen to correspond to SNPs in locations with functional effects, thus altering overall redox balance for the initiation and development of ovarian cancer. Future studies examining such SNPs are important to fully elucidate a genotype switch mechanism induced by talc exposure.

In summary, this research clearly demonstrates that talcum powder induces inflammation and alters the redox balance favoring a pro-oxidant state in normal and EOC cells. This study has shown a dose-dependent significant increase in key pro-oxidants, iNOS, $NO2^-$/$NO3^-$, and MPO and a concomitant decrease in key antioxidant enzymes, CAT, SOD, GPX, and GSR, in all talc treated cells (both normal and ovarian cancer) compared to their controls. Additionally, there was a significant increase in CA-125 levels in all the talc treated cells compared to their controls, except in macrophages (which do not produce CA-125). The mechanism by which talc alters the cellular redox and inflammatory balance involves the induction of specific mutations in key oxidant and antioxidant enzymes that correlate with alterations in their activities. The fact that these mutations

happen to correspond to known SNPs of these enzymes indicate a genetic predisposition to developing ovarian cancer with genital talcum powder exposure.

Summary of opinions

These opinions are made to a reasonable degree of scientific certainty and are based on my experience, training, and expertise, as well as a knowledge of the relevant scientific literature and my previous and ongoing research.

1. Johnson's Baby Powder elicits an inflammatory response in normal ovarian and tubal cells and in ovarian cancer cells that can result in the development and the progression of ovarian cancer.
2. This pro-carcinogenic process involves oxidative stress, alteration of the redox environment by increasing oxidant enzymes and decreasing anti-oxidant enzymes, promotion of cell proliferation, inhibition of apoptosis, and induction of specific genetic mutations.
3. Johnson's Baby Powder exposure results in elevation of CA-125, a clinically relevant biomarker for ovarian cancer, in normal and ovarian cancer cells.
4. The molecular effects resulting from Johnson's Baby Powder exposure exhibit a clear dose-response pattern.
5. In my opinion, based on established molecular mechanisms for the pathogenesis of ovarian cancer (as evidenced in the peer-reviewed scientific literature and my previously published research) and my *in vitro* experiments, Johnson's Baby Powder exposure can cause ovarian cancer.
6. In my opinion, based on established molecular mechanisms that influence the progression and chemoresistance associated with ovarian cancer (as evidenced in the peer-reviewed

scientific literature and my previously published research) and my *in vitro* experiments, Johnson's Baby Powder exposure worsens the prognosis for patients with ovarian cancer.

I reserve the right to amend or supplement this report as new information becomes available.

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Exhibit A

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EDUCATION:

Ph.D. in Molecular Biology University of Essex, Colchester, England, United Kingdom	1983–1986
B.S. in Biochemistry King Saud University, Riyadh, Saudi Arabia	1979–1982

POSTGRADUATE TRAINING:

Fellowship in Immunopathology, University of Michigan, Ann Arbor, MI	1992–1993
Fellowship in Molecular Biology, Henry Ford Hospital, Detroit, MI	1988–1990

FACULTY APPOINTMENTS:

Adjunct Associate Professor, Department of Oncology, Karmanos Cancer Institute, Detroit Medical Center/Wayne State University School of Medicine, Detroit, MI	2017–Present
Director, Ovarian Cancer Biology Research, Department of Obstetrics and Gynecology, Wayne State University School of Medicine, Detroit, MI	2009–Present
Scientific Member, Karmanos Cancer Institute, Molecular Biology and Genetics Program, Wayne State University School of Medicine, Detroit, MI	2008–Present
Member of Tumor Biology and Microenvironment Program, Karmanos Cancer Institute, Detroit, MI	2007–Present
Associate Professor (secondary), Department of Physiology, Wayne State University School of Medicine, Detroit, MI	2008–Present
Associate Professor (primary), Department of Obstetrics and Gynecology,	2007–Present

Wayne State University School of Medicine, Detroit, MI

Tenure, Department of Obstetrics and Gynecology, Wayne State University School of Medicine, Detroit, MI	2007–Present
Associate Status, Department of Anatomy/Cell Biology, Wayne State University School of Medicine, Detroit, MI	2003–Present
Tenure Track, Department of Obstetrics and Gynecology, Wayne State University School of Medicine, Detroit, MI	2001–2007
Assistant Professor, Department of Obstetrics and Gynecology, Wayne State University School of Medicine, Detroit, MI	1998–2007

HOSPITAL OR OTHER PROFESSIONAL:

Senior Investigator, Center for Biomedical Research, Oakland University, Rochester, MI	1997–1998
Adjunct Associate Professor, Department of Chemistry, Oakland University, Rochester, MI	1996–2004
Bioscientific Staff Investigator, Dermatology Department, Henry Ford Hospital, Detroit, MI	1995–1998
Associate Staff Investigator, Department of Dermatology, Henry Ford Hospital, Detroit, MI	1993–1994
Special Lecturer, Department of Chemistry, Oakland University, Rochester, MI	1991–1996
Assistant Staff Investigator, Hypertension Research, Henry Ford Hospital, Detroit, MI	1990–1992
Technical Manager, Pharmaceutical Company, Riyadh, Saudi Arabia	1987–1988
Research Assistant, Biochemistry Department, King Saud University, Riyadh, Saudi Arabia	1982–1983

MAJOR PROFESSIONAL SOCIETIES

Associate Member, Society for Gynecologic Oncology	2017–Present
Member, American Association of Cancer Research	2008–Present
Member, American Federation of Clinical Research	2009–Present
Member, American Society for Reproductive Medicine	1998–Present
Member, Society for Reproductive Investigation	1998–Present
Member, American Association of University Professors	1996–Present
Member, American Chemical Society	1991–2014
National Research Council of the United Kingdom	1985–1986
Medical Research Council of the United Kingdom	1984–1998

HONORS/AWARDS:

Star Award 2017
73rd American Society for Reproductive Medicine (ASRM) Scientific Congress & Expo
This award recognizes members who have presented during at least nine of the ASRM Annual Meetings from the years 2007-2016. Presentations may include Congress courses and/or seminars, Scientific Program symposia, posters, and/or oral presentation of abstracts. 234 awardees

Award for Research 2017

Awarded to Nicole King, PhD, Postdoctoral Fellow in the laboratory of Dr. Ghassan Saed
73rd American Society for Reproductive Medicine (ASRM Scientific Congress & Expo
The purpose of this award is to recognize outstanding research conducted by individuals in training under the "Reproductive Surgery" category. He/she is a presenting first author, and a medical student, resident, fellow, or under-graduate, graduate, or postdoctoral student. Three awardees

Star Award 2016
72nd American Society for Reproductive Medicine (ASRM) Scientific Congress & Expo
This award recognizes members who have presented during at least nine of the ASRM Annual Meetings from the years 2007-2015. Presentations may include Congress courses and or seminars, Scientific Program symposia, posters, and/or oral presentation of abstracts. ~235 awardees

Excellence in Biomedical Research 2015
Global Medical Discovery Series
Key Scientific Article for peer-reviewed publication entitled: "Sox2 Gene Amplification Significantly Impacts Overall Survival in Serous Epithelial Ovarian Cancer." Reproductive Sciences 22(1):38-46, 2015. Epub July 18, 2014. One awardee

Star Award 2013
69th American Society for Reproductive Medicine Annual Meeting (ASRM)
This award recognizes members who have presented during at least nine of the ASRM Annual Meetings from the years 2007-2012. Presentations may include Congress courses and/or seminars, Scientific Program symposia, posters, and/or oral presentation of abstracts. ~235 awardees

Star Award 2011
67th American Society for Reproductive Medicine (ASRM) Annual Meeting
This award recognizes members who have presented during at least nine Of the ASRM Annual Meetings from the years 2007-2010. Presentations may include Congress courses and/or seminars, Scientific Program symposia, posters, and/or oral presentation of abstracts. ~235 awardees

President's Award for Excellence in Teaching 2009
Wayne State University School of Medicine
This award is in recognition for outstanding faculty who have made contributions to teaching at WSU to an exceptionally high degree, demonstrate comprehensive knowledge of their subject, superior classroom performance, and high educational standards; communicate their subject matter accurately, clearly, and effectively; generate enthusiasm and respect for learning; motivate their students to excel; and are accessible to students; innovative instructional practices, impact on teaching at WSU, and contributions to advancing teaching in their field.

Finalist Paper 2006
62nd American Society for Reproductive Medicine (ASRM) Annual Meeting
One awardee

Prize Paper Candidate 2005
Conjoint 61st American Society for Reproductive Medicine (ASRM) Annual Meeting and 51st Canadian Fertility and Andrology Society Annual Meeting
One awardee

Finalist Paper 2005
61st American Society for Reproductive Medicine (ASRM) Annual Meeting
One awardee

Finalist Paper 2003
Society of Reproductive Endocrinology and Infertility (SREI) Annual Meeting
One awardee

Finalist Paper, Basic Science 2003
19th European Society of Human Reproduction and Embryology (ESHRE) Annual Meeting
One awardee

Award Paper 2000
58th Society of Reproductive Surgeons (SRS) Scientific Program

Finalist Paper 2000
Society of Reproductive Endocrinology and Infertility (SREI) Annual Meeting
One awardee

Award Paper 1998
52nd Society of Reproductive Surgeons (SRS) Scientific Program

Outstanding Professor of the Year Award 1996–1997
Golden Key National Society, Oakland University Chapter, Rochester, MI
One awardee

SERVICE:

Wayne State University

Departmental/Divisional

Chairperson, Organizing Committee, 2017 Joint Annual Reproductive Sciences Retreat, Departments of Obstetrics and Gynecology, Wayne State University School of Medicine and University of Toronto; and The Michigan Alliance for Reproductive Technologies and Sciences (MARTS) Annual Meeting at Wayne State University	2017
Faculty Mentor, NIH/NICHD Women's Reproductive Health Research (WRHR) Scholar Program, Department of Obstetrics and Gynecology	2012–2016
Faculty Associate, Fulbright Visiting Senior Scholar Award recipient Dr. Iyad Ali, Department of Obstetrics and Gynecology	2012–2014
Member, Selective Salary Committee, Department of Obstetrics and Gynecology	2012–Present
Member, Promotion and Tenure Committee, Department of Obstetrics and Gynecology	2012–Present
Chairperson, C.S. Mott Center Seminar Series Committee, Department of Obstetrics and Gynecology	1998–Present
Chairperson, Basic Research Endocrine Fellows Training Committee, Department of Obstetrics and Gynecology	1998–2014
Member, Reproductive Endocrinology and Infertility Fellowship Selection Committee, Department of Obstetrics and Gynecology	1998–Present

School of Medicine

Member, Strategic Research Initiative Grant Review (SRIG) Committee, Karmanos Cancer Institute	2013–2014
Member, PhD Committee for Batoul Abdullah, PhD Candidate, Center for Molecular Medicine and Genetics, Wayne State University	2012–2016
Member, PhD Committee for Jimmy Belotte, MD, PhD Candidate, Department of Physiology and Reproductive Sciences, Department of Obstetrics and Gynecology, Wayne State University	2012–2016
Faculty, Reproductive Sciences Graduate Program, Department of Physiology, Wayne State University	2012–2016
Member, Search Committee for a candidate selection for a joint appointment in Departments of Psychology and Obstetrics & Gynecology in the field of Psychopharmacology	2003–2005

Affiliate Medical Organizations

Member, Karmanos Cancer Institute, Molecular Biology and Genetics Program, Detroit, MI	2008–Present
Member of Tumor Biology and Microenvironment Program, Karmanos Cancer Institute, Detroit, MI	2007–Present

Professional

President, National Arab American Medical Association, Michigan Chapter, Troy, MI	2017–Present
Judge, American Society for Reproductive Medicine (ASRM) Abstract Selection Committee, Birmingham, AL	2015–Present
President, National Arab American Medical Association, Michigan Chapter, Troy, MI	2014–2015
Board Member, National Arab American Medical Association, Michigan Chapter, Troy, MI	2009–Present
Judge, Poster Finals Prize Committee, 2002 Annual Meeting of the American Society for Reproductive Medicine (ASRM), Birmingham, AL	2002–2003

Community

Biohazard and Safety Committee, Henry Ford Hospital, Detroit, MI	1996–1998
Radioisotope Safety Committee, Henry Ford Hospital, Detroit, MI	1996–1998
Journal Club Committee, Department of Dermatology, Henry Ford Hospital, Detroit, MI	1994–1998
Basic Research Training Committee, Department of Dermatology, Henry Ford Hospital, Detroit, MI	1994–1998
Animal Care Committee, Henry Ford Hospital, Detroit, MI	1993–1996

Consulting

Consultant, Molecular Biologic Testing, DS Biotech, Detroit, MI	2013–Present
Consultant, Application of Cyclooxgenase-2 in the Treatment of Ovarian Cancer, Pfizer Pharmaceuticals, Rochester, MI	2002
Consultant, Technical expertise in developing molecular probes and markers, Oxford Biomedical Research, Oxford, MI	1991–1998

Scholarly Service

<u>Grant Review Committees</u>

Member, Scientific Review Committee, Ethel F. Donoghue Women's Health Investigator Program, Yale University, New Haven, CT	2004

Service for Peer-Reviewed Journals Editorship

Editorial Board Membership:

Editor-in-Chief, Gynecology and Obstetrics Research-Open Journal	2015–Present

Review of Manuscripts and Chapters:

Journal of Cellular and Molecular Medicine	2015–Present
Systems Biology in Reproductive Medicine	2013–Present
Journal of Assisted Reproduction and Genetics	2013–Present
Journal of Reproductive Science	2012–Present
European Journal of Obstetrics & Gynecology and Reproductive Biology	2009
Gastroenterology	2007
Houghton Mifflin Company, College Division	2003
American Gynecological and Obstetrical Society	2003
Oncogenes	2003
Fertility and Sterility	2001–Present
Wound Repair and Regeneration	2000–Present
Journal of Cytokine Research	1998–2000

TEACHING

Teaching at Wayne State University

Undergraduate Students

Instructor. Department of Biological Sciences – BIO 3990: Undergraduate course primarily for biology majors who wish to continue in a field beyond that covered in regular courses under the direction of Biological Sciences faculty.

Instructor and Advisor. Department of Physiology – PSL 5010: Undergraduate course involving student participation in laboratory research in the physiological sciences under the supervision of a departmental faculty advisor.
This course involves an introduction to experimental protocol and current related scientific literature.

Advisor. Department of Biological Sciences – BIO 6990: Undergraduate course for honors students involving student participation in laboratory research in the physiological sciences under the supervision of a departmental faculty advisor.

Graduate Students

Instructor. Interdisciplinary Biomedical Sciences – IBS 7060: Biomedical Endocrine and Reproductive Systems Development.
This course is for graduate students within the Ph.D. Program in Anatomy and Cell Biology of which has the aim of providing a broad based knowledge of the important areas of biomedical research.

Instructor. Department of Physiology with Concentration in the Reproductive Sciences Program (PhD) – RPS 7350: Biomolecular Techniques: From Genes to Protein

Instructor. Department of Physiology with Concentration in the Reproductive Sciences Program (PhD), Principles of Reproductive Biology – PSL 7690: Cancers in Reproductive Organs/ Journal Club.
This lecture explains the impact of cancer in women; to discuss the epidemiology, risk factors, screening modalities and preventative strategies of gynecologic cancers and the role of stem cells.

Instructor. Current Research Topics in the Reproductive Sciences – PSL 7775: Molecular Mechanisms of Postoperative Adhesions.
This course is for graduate students within the Ph.D. Program in Physiology with Concentration in the Reproductive Sciences of which covers current research topics in reproductive sciences. The Program itself incorporates the teaching, research and physical resources of both the Physiology and the Obstetrics and Gynecology Departments, offering interdisciplinary doctoral training in a clinical environment in the reproductive sciences. The primary academic focus engages teaching and research training in reproduction and development, with an emphasis on the following: developmental biology, perinatal biology, reproductive endocrinology, reproductive genetics, toxicology/teratology and molecular biology including genomics, proteomics, and bioinformatics. Dissertation research is under the mentorship of Obstetrics and Gynecology basic science graduate faculty.

Advisor and Mentor. Current Research Topics in the Reproductive Sciences – PSL 7996: Arranged Research.
This course is for the graduate students within the "Ph.D. Program in Physiology with Concentration in the Reproductive Sciences" (as described in PSL 7775) which covers graduate level experiences in research techniques. It is required that special research topics, within specified areas, be agreed upon between individual faculty members and students.

Advisor and Mentor. Doctoral Candidate Status I-IV – PSL 9991, 9992, 9993, 9994: Thesis/Dissertation Research and Design.
This course is for the graduate students within the "Ph.D. Program in Physiology with Concentration in the Reproductive Sciences" (as described in PSL 7775). Required in consecutive academic-year semesters following advancement to Ph.D. candidacy status I through IV.

Advisor and Mentor. Doctoral Candidate Dissertation Research and Direction – PSL 9995: Candidate Maintenance Status.
This course is for the graduate students within the "Ph.D. Program in Physiology with Concentration in the Reproductive Sciences" as described above in PSL 7775. Required after completion of 30 credits in PSL 9991-9994.

Director. Summer Reproductive Technology Course. The CS Mott Center for Human Growth and Development, Division of Reproductive Endocrinology and Infertility, Department of Obstetrics and Gynecology, 2014-2015.
The course design is to allow for Reproductive Endocrinology and Infertility/Medical Genetics fellows, as well as graduate students to become familiar with laboratory techniques in the reproductive sciences. The graduate students will acquire a thorough understanding of the

theory and special methodology utilized to perform techniques indicative of reproductive endocrinology and infertility.

Lecturer. Laboratory Techniques. Summer Reproductive Technology Course. The CS Mott Center for Human Growth and Development, Division of Reproductive Endocrinology and Infertility, Department of Obstetrics and Gynecology, 2014.
This lecture explains the various laboratory techniques, and their limitations, as applied to the reproductive sciences.

Lecturer. Molecular Biological Procedures. Summer Reproductive Technology Course. The CS Mott Center for Human Growth and Development, Division of Reproductive Endocrinology and Infertility, Department of Obstetrics and Gynecology, 2015.
This lecture explains the various laboratory techniques, and their limitations, as applied to the reproductive sciences.

Residents and Fellows

Director. Summer Reproductive Technology Course. The CS Mott Center for Human Growth and Development, Division of Reproductive Endocrinology and Infertility, Department of Obstetrics and Gynecology, 2014-2015.
The course design is to allow for Reproductive Endocrinology and Infertility/Medical Genetics fellows, as well as graduate students to become familiar with laboratory techniques in the reproductive sciences. The fellows will acquire a thorough understanding of the theory and special methodology utilized to perform techniques indicative of reproductive endocrinology and infertility.

Instructor. PCR Technique: Concept and Clinical Application Course. The CS Mott Center for Human Growth and Development, Division of Reproductive Endocrinology and Infertility, Department of Obstetrics and Gynecology, 2012-Present.
This course is designed to allow departmental residents, fellows, Reproductive Endocrinology and Infertility/Medical Genetics fellows, and interested graduate students (within the C.S. Mott Center) to become familiar with the PCR technique and how to use it effectively within the laboratory.

Teaching at Other Institutions

Undergraduate Students

Adjunct Associate Professor. Taught two undergraduate courses, CHM104 “Introduction to Chemical Principles” and CHM201 “Introduction to Organic and Biological Chemistry” for nursing and health sciences students at the Department of Chemistry, Oakland University, Rochester, MI, 1991-2004.

Graduate Students

Instructor. Four-day workshop: PCR Techniques, Concepts and Applications. Howard Hughes Research Program, Oakland University, Rochester, MI, May 19-22, 1998.
This workshop was for graduate, postdoctoral, laboratory research personnel, and faculty within the field of science and research.

Instructor. Taught a graduate course CHM554 “Molecular Biology and Biotechnology” at the Department of Chemistry, Oakland University, Rochester, MI, 1995-1998.

Instructor. Biotechnology: From Genes to Proteins. Department of Dermatology, Oakland University, Rochester, MI, 1993-1998.
This course was part of the Research Training in Biotechnology Program postgraduate curriculum for residents and fellows to utilize state-of-the-art molecular technology techniques to answer questions related to molecular pathogenesis of skin diseases such as skin cancer, fibrosis and wound healing.

Teaching Assistant. Introduction to Chemical Principals. Department of Chemistry, University of Essex, Colchester, England, United Kingdom, 1987-1988.

Residents and Fellows

Instructor and Laboratory Advisor. Biotechnology Research Training. Department of Dermatology, Oakland University, Rochester, MI, 1993-1998.
This program trained dermatology residents to utilize state-of-the-art molecular technology techniques to answer questions related to molecular pathogenesis of skin diseases such as skin cancer, fibrosis and wound healing.

Mentorship

Mentor on research projects related to endometriosis, postoperative adhesions, and ovarian cancer to the Department of Obstetrics and Gynecology past and present undergraduate and graduate students, residents, clinical and postgraduate fellows, scholars, faculty, and research technicians, assistants and associates.

Undergraduate Students:

Yousif Abbiss; Newaj Abdullah; Dana Abufarha, Shadi Abuolba Ahmad [awarded the 2007 Wayne State University School of Medicine Undergraduate Research Scholarship Award]; Ali Alarab; Radi Al-Dasouqi; Danna Al-Hadidi; Jeremy Berman; Chelsea Fortin; Ellory Greenberg; Waseem Imann; Shucni Jain; Marisa Karcz; Hadil Katato; Reem Khazaal; Yanamandra Krishnakant;, Wasfeh Musheinish; Bailey Neubauer; Osama Nusrat; Tessy Oommen; Norman Orabi; Alex Papadellis; Sonica Rehan; James Waleke [WSU School of Medicine 2004 graduate]; Rani Yaldo, Yousif Younan; Nabaa Zalzala; and Xuping (Sherry) Zhu.

Graduate:

Osama Nusrat, MD (Master/past): Nusrat O, Belotte J, Fletcher NM, Memaj I, Saed MG, Diamond MP, **Saed GM**. The role of angiogenesis in the persistence of chemoresistance in epithelial ovarian cancer. Reproductive Sciences 23(11):1484-1492, 2016. PMID: 27122375

Batoul Abdullah, PhD: Abdallah BY, Horne SD, Stevens JB, Liu G, Ying AY, Vanderhyden B, Krawetz SK, Gorelick R, Heng HH (2013). Single cell heterogeneity: Why unstable genomes are incompatible with average profiles. Cell Cycle 12:3640-3649, 2013. PMID: 24091732 PMCID: PMC3903715

Jimmy Belotte, MD, PhD: Belotte J, Fletcher NM, Saed MG, Abusamaan MS, Dyson G, Diamond MP, **Saed GM**. A single nucleotide polymorphism in catalase is strongly associated

with ovarian cancer survival. PLoS One 10(8):e0135739, 2015. eCollection 2015. PMID: 26301412 PMCID: PMC4547699

Nicole Fletcher-King, PhD: Fletcher NM, Belotte J, Saed MG, Memaj I, Diamond MP, Morris RT, **Saed GM**. Specific point mutations in key redox enzymes are associated with chemoresistance in epithelial ovarian cancer. Free Radical Biology and Medicine 102:122-132, 2017. PMID: 27890641

Jennell White, PhD: White JC, Jiang ZL, Diamond MP, **Saed GM**. Macrophages induce the adhesion phenotype in normal peritoneal fibroblasts. Fertility and Sterility 96(3):758-763.e3, 2011. Epub July 27, 2011. PMID: 21794857

Residents

Drs. (MD) Zeynep Alpay, Dana Ambler, Tarek Dbouk, Eslam Elhammady, and Valerie Shavell.

Clinical and Postgraduate Fellows:

Drs. (MD) Mazen Abdallah, Awoniyi Awonuga [faculty], Jashoma Banerjee, Alan Bolnick, Jay Bolnick, Subodhsingh Chauhan, Laura Detti, Michael Freeman, April Gago, Roohi Jeelani, Sana Khan, Mohamed Mitwally, Valerie Shavell, Mili Thakur, Rahi Victory, and Terri Woodard; and Christopher Bryant, MD, Associate Professor, Division of Gynecologic Oncology (past faculty).

Of note, the aforementioned have participated in premier annual scientific meetings of the American Society for Reproductive Medicine, Society for Free Radical Biology and Medicine, Society for Reproductive Investigation, and American College of Obstetrics and Gynecology, just to name a few, as well as publishing their many scientific achievements (articles and abstracts) in preeminent peer-reviewed journals (see Publications section).
Acknowledgement: Michael Freeman, MD [past fellow], was awarded a $20,000 research grant from the American Gynecologic and Obstetrical Society (AGOS) during his fellowship [1999-2002]. Alan Bolnick, MD and Sana Khan, MD [past fellows, 2013-2016] were each awarded from the Pacific Coast Reproductive Society, the 2015 Travel Award, as well as Roohi Jeelani, MD and Mili Thakur, MD [past fellows, 2015-2017 and 2014-2017, respectively] who were each awarded the 2016 Travel Award. These AGOS travel awards paid for registration to the annual meeting, course fees, and all travel expenses incurred.

Lastly, Mili Thakur, MD [past fellow, 2014-2017] of the combined Reproductive Endocrinology and Infertility and Medical Genetics Fellowship program (the only one of its kind in the country), was the recipient of the 2016 Pfizer-SRI (Society for Reproductive Investigation), President's Presenter's Award. This award for given to Mili for her abstract entitled, "*Galactose and Its Metabolites Deteriorate Metaphase II Mouse Oocyte Quality through a Mechanism that Involves the Generation of Reactive Oxidative Species, Mitochondrial Dysfunction and Apoptosis*." The President's Presenter's Award is given in recognition of the 25 most meritorious abstracts (either poster or oral presentation) submitted by individuals still in training. Dr. Thakur received this prestigious award at the 63rd Annual Meeting of the Society for Reproductive Investigation, Montreal, Quebec, Canada, in March of 2016.
Scholars

Iyad Ali, PhD: Assistant Professor of Biochemistry, Faculty of Medicine and Health Sciences, An-Najah National University, Nablus, Palestine; visiting Fulbright Arab Fund Fellowship Scholar in the laboratories of Drs. Husam Abu-Soud and Ghassan Saed, Division of Reproductive

Endocrinology and Infertility, Department of Obstetrics and Gynecology, Wayne State University School of Medicine.

Awoniyi Awonuga, MD: Associate Professor, Women's Reproductive Health Research (WRHR) Scholar, Division of Reproductive Endocrinology and Infertility, Department of Obstetrics and Gynecology, Wayne State University School of Medicine. Citation: Awonuga AO, Belotte J, Abuanzeh S, Fletcher NM, Diamond MP, **Saed GM**. Advances in the pathogenesis of adhesion development: the role of oxidative stress. Reproductive Sciences 21(7):823-836, 2014. Epub February 11, 2014. Review. PMID: 24520085 PMCID: PMC4107571

Jimmy Belotte, MD, PhD: Assistant Professor, Women's Reproductive Health Research (WRHR) Scholar, Division of Reproductive Endocrinology and Infertility, Department of Obstetrics and Gynecology, Wayne State University School of Medicine. Citation: *Belotte J, *Fletcher NM, *Alexis M, Morris RT, Munkarah AR, Diamond MP, **Saed GM**. Sox2 gene amplification significantly influences overall survival in serous epithelial ovarian cancer. Reproductive Sciences 22(1):38-46, 2015. Epub July 18, 2014. PMID: 25038052 PMCID: PMC4275450

Lylia Fahmy, MD: Clinical Instructor, Women's Reproductive Health Research (WRHR) Scholar, Division of Reproductive Endocrinology and Infertility, Department of Obstetrics and Gynecology, Wayne State University School of Medicine. Thesis: Effect of Ovarian Hormones on Adhesion Development. 2005.

Faculty

Mentor of current and past Obstetrics and Gynecology clinical faculty through collaborations on research projects and grant submissions. Faculty members are as follows: Awoniyi Awonuga, MD, Professor, Division of Reproductive Endocrinology and Infertility and Women's Reproductive Health Research (WRHR) Scholar (training completed December 2015); Jimmy Belotte, MD, Associate Professor, Division of Gynecology, Women's Reproductive Health Research (WRHR) Scholar, (training completed September 2016 with PhD); Lylia Fahmy, MD, past Clinical Instructor, Division of Reproductive Endocrinology and Infertility, Women's Reproductive Health Research (WRHR) Scholar, (training completed 2005); and Peter Baumann, MD, Associate Professor, Division of Gynecology (retired).

I have also been instrumental to key professional presentations at local, national, and international conferences by our past and present senior faculty members of the Obstetrics and Gynecology Department. They are as follows: Adnan Munkarah, MD, Professor and Director, Division of Gynecologic Oncology; Bernard Gonik, MD, Professor, Division of Maternal and Fetal Medicine; Jay Berman, MD, Associate Professor and Associate Chair, Department of Obstetrics and Gynecology and Director, Division of Gynecology; John Malone, Jr, MD, Professor and past Chair, Department of Obstetrics and Gynecology (deceased); Kamran Moghissi, MD, Professor Emeritus, past Chair Emeritus, Department of Obstetrics and Gynecology, past Director, Division of Reproductive Endocrinology and Infertility, and past Director, CS Mott Center for Human Growth and Development (retired); and Michael Diamond, MD, Professor, past Associate Chair, Department of Obstetrics and Gynecology, past Director, Division of Reproductive Endocrinology and Infertility, and past Assistant Dean of Clinical and Translational Research, Wayne State University School of Medicine (now at Georgia Regents University, Augusta, GA).

Research Associates/Assistants/Technicians

In the laboratory of Dr. Ghassan Saed: Drs. (PhD) Boytcho Boytchev, Semira Galijasevic, Zhongliang (John) Jiang, MD, Hong Lu, Qui Lu, Gheorghe Proteasa, Natalie Rizk, Rona Wang, MD, and Ming Zhao, MD; Danielle Hall, BS, Nicole Fletcher-King, BS, MS, and Manal Omar, BS.

Essays/Theses/Dissertations Directed

Name: Osama Nusrat, MD (Master Degree/past), Department of Physiology and Reproductive Sciences (2015-2017), Wayne State University School of Medicine, Detroit, MI.

Dissertation Title: The Role of Angiogenesis in the Persistence of Chemoresistance in Epithelial Ovarian Cancer

Date Awarded: September 2017

Current Status: Resident, Department of Internal Medicine, University of Arizona College of Medicine, Tucson, AZ

Name: Jimmy Belotte, MD, PhD, Department of Physiology in the Reproductive Sciences Concentration (2012-2016); and Women's Reproductive Health Research (WRHR) Scholar, Department of Obstetrics and Gynecology, Wayne State University School of Medicine, Detroit, MI

Dissertation Title: The Role of Oxidative Stress in the Establishment of Resistance to Cisplatin in Epithelial Ovarian Cancer Cells

Date Awarded: September 14, 2016 and WRHR training completed September 14, 2016

Current Status: Associate Professor, Division of Gynecology, Department of Obstetrics and Gynecology, Wayne State University School of Medicine, Detroit, MI

Name: Batoul Abdullah, PhD, Department of Physiology in the Center for Molecular Medicine and Genetics Concentration (2012-2016), Wayne State University School of Medicine, Detroit, MI

Dissertation Title: Fuzzy Inheritance: A Novel Form of Somatic Cell Inheritance that Regulates Cell Population Heterogeneity

Date Awarded: 2016

Current Status: Postdoctoral Fellow in the laboratory of Henry (Hong-Qiang) Heng, PhD, Center for Molecular Medicine & Genetics and Pathology, Wayne State University School of Medicine, Detroit, MI

Name: Awoniyi Awonuga, MD, Women's Reproductive Health Research (WRHR) Scholar, Department of Obstetrics and Gynecology, Wayne State University School of Medicine, Detroit, MI

Thesis Title: Oxidative Stress in the Pathogenesis of Post-Operative Adhesions

Training Completed: December 2015

Current Status:	Professor and Interim Director, Division of Reproductive Endocrinology and Infertility, Department of Obstetrics and Gynecology, Wayne State University School of Medicine, Detroit, MI
Name:	Nicole Fletcher-King, PhD, Department of Physiology in the Reproductive Sciences Concentration Program (2008-2013), Department of Obstetrics and Gynecology, Wayne State University School of Medicine, Detroit, MI
Dissertation Title:	The Role of Oxidative Stress in the Pathogenesis of Epithelial Ovarian Cancer
Date Awarded:	September 2013
Current Status:	Postdoctoral Fellow in the laboratory of Ghassan M Saed, PhD, Division of Reproductive Endocrinology and Infertility, Department of Obstetrics and Gynecology, CS Mott Center for Human Growth and Development, Wayne State University School of Medicine, Detroit, MI
Name:	Jennell White, PhD, Department of Physiology in the Reproductive Sciences Concentration Program (2000-2011), Department of Obstetrics and Gynecology, Wayne State University School of Medicine, Detroit, MI
Dissertation Title:	The Potential Role of Innate Immunity in the Pathogenesis of Postoperative Adhesions
Date Awarded:	September 2011
Current Status:	Postdoctoral Fellow, Department of Pediatrics, Wayne State University School of Medicine, Detroit, MI
Name:	Lylia Fahmy, MD, Women's Reproductive Health Research (WRHR) Scholar, Department of Obstetrics and Gynecology, Wayne State University School of Medicine, Detroit, MI
Thesis Title:	Effect of Ovarian Hormones on Adhesion Development
Date Completed:	September 2005
Current Status:	Associate Professor, Department of Obstetrics and Gynecology, University of Nebraska Medical Center, Omaha, NB

Course or Curriculum Development

Originator and Director. Summer Reproductive Technology Course. 2014
This course design is to allow for Reproductive Endocrinology and Infertility/Medical Genetics fellows, as well as graduate students, to become

familiar with all aspects of laboratory techniques within the field of reproductive sciences. Division of Reproductive Endocrinology and Infertility, Department of Obstetrics and Gynecology, The C.S. Mott Center for Human Growth and Development, Wayne State University School of Medicine, Detroit, MI.

Course Director. Reproductive Sciences Concentration – RPS 7350: Biomolecular Techniques: From Genes to Protein. 2006
This course design is specifically for graduate students enrolled in the PhD Program in Physiology with Concentration in the Reproductive Sciences, as part of their curriculum. This is an integrated PhD program incorporating the teaching, research, and physical resources of two departments -- Physiology and Obstetrics & Gynecology at Wayne State University School of Medicine, Detroit, MI.

Organizer. Four-day workshop (May 19-22): PCR Techniques, Concepts, and Applications. 1998
Workshop developed for undergraduates, graduates, postdoctoral, laboratory personnel, and faculty studying and/or working within the field of science and research. Sponsored by the Howard Hughes Research Program of Oakland University, Rochester, MI.

Designer. Introduction to Molecular Cloning. 1996
Course designed to teach techniques for characterization and manipulation of DNA and RNA from the basis of modern biomedical research. Coursework pertinent towards medical residents and fellows at the Henry Ford Hospital, Detroit, MI, and graduate students at Oakland University, Rochester, MI.

Designer. Research Training in Biotechnology. 1993
This program trained Department of Dermatology residents and fellows to utilize state-of-the-art molecular technology techniques to answer questions related to molecular pathogenesis of skin diseases such as skin cancer, fibrosis and wound healing at Henry Ford Hospital, Detroit. MI. This training ended in 1998.

Course Director. I have participated in developing the course, Introduction to Chemical Principles (CHM 104) to meet general education requirements. CHM 104 satisfies the university general education requirement in natural science and technology (NST). The learning outcomes for NST courses state that the student will demonstrate knowledge of major concepts from natural science or technology, including developing and testing of hypotheses, drawing conclusions, and reporting of findings through some laboratory experience or an effective substitute. This course taught at Oakland University, Rochester, MI. 1991–2004

Designer. Laboratory course. I was actively involved in developing and instructing two laboratory sections for CHM 104. Students learned how to evaluate sources of information in science or technology. Developed at Oakland University, Rochester, MI 1991–2004

Designer. I developed and taught CHM 104 and CHM 201 to nursing students 2005–2010

on-line (a web-based instruction). I designed courses to satisfy the university general education requirement in natural science and technology (NST). For this, I utilized and implemented the virtual chemistry laboratory experience to be an integral part of this course. Developed at Oakland University, Rochester, MI,

GRANTS, CONTRACTS, AND OTHER FUNDING:

Active National/International Grants and Contracts

Role: Mentor, Percent Effort: 0%; Principal Investigator: NM King, PhD
Title: "Novel Biomarkers for Early Detection of Ovarian Cancer." The project's design is to identify key markers of oxidative stress that have the potential to serve as screening tools for ovarian cancer and may play a role in the acquisition of chemoresistance.
Source: Prevent Cancer Foundation, Postdoctoral Fellowship Grant
Date: 01/01/16 – 12/31/18
Total Direct Costs: $80,000

Role: Principal Investigator, Percent Effort: 5%
Title: "Elucidation of Cellular Mechanisms of Evitar of Post-Operative Fibrosis."
Source: Temple Therapeutics, 25S8P1
Date: 04/01/17 – 05/31/18
Total Direct Costs: $100,000

Pending National/International Grants and Contracts

Role: Mentor, Percent Effort: 0%; Principal Investigator: NM King, PhD
Title: "Novel Mechanism of Apoptosis in Chemoresistant Ovarian Cancer Cells." To determine whether chenoresistance in ovarian cancer manifests decreased apoptosis through enhanced s-nitrosylation of caspase-3 mechanism which can, thereby, be reversed by DCA.
Source: American Association for Cancer Research (AACR)
Date: 07/01/17 – 06/30/19
Total Direct Costs: $100,000

Role: Principal Investigator, Percent Effort: 30%
Title: "Identification of a Novel Target with Intriguing Anti-Tumorigenic Effects in Ovarian Cancer." To identify and test a target that cross-reacts with the CD11b antibody and determines its efficacy in killing both sensitive and chemoresistant ovarian cancer cells.
Source: NIH/NICHD R01, Proposal #17-0220
Date: 09/01/17 – 08/31/22
Total Direct Costs: $1,919,600

Role: Mentor, Percent Effort: 0%; Principal Investigator: NM King, PhD
Title: "Novel Marker of Survival in Ovarian Cancer Cells." To test the anti-tumorigenic potential of integrin αV/β1 antibodies in sensitive and chemoresistant ovarian cancer.
Source: U.S. Department of Defense (DOD)
Date: 01/01/18 – 12/31/19
Total Direct Costs: $385,000

Role: Principal Investigator, Percent Effort: 25%
Title: "Cross-Talk Between MPO and iNOS Regulates Apoptosis in Chemoresistant Ovarian Cancer." To determine whether chemoresistance in ovarian cancer manifests decreased apoptosis through enhanced s-nitrosylation of caspase-3 mechanism, which can be reversed by DCA.
Source: NIH/NICHD R21
Date: 09/01/17 – 08/31/19
Total Direct Costs: $423,500

Role: Principal Investigator, Percent Effort: 10%
Title: "A Novel Target with Intriguing Anti-Tumorigenic Effects in Cancer." To identify and test a target that cross-reacts with the CD11b antibody and determines its efficacy in killing both sensitive and chemoresistant ovarian cancer cells.
Source: NIH/DHHS Small Business Technology Transfer Grant (STTR), R41
Date: 07/01/17 – 6/30/18
Total Direct Costs: $299,999

Role: Principal Investigator, Percent Effort: NA
Title: "A Novel Target with Intriguing Anti-Tumorigenic Effects in Cancer." To identify and test a target that cross-reacts with the CD11b antibody and determines its efficacy in killing both sensitive and chemoresistant ovarian cancer cells.
Source: The Honorable Tina Brozman Foundation, Inc. for Ovarian Cancer Research – Letter of Intent
Date: 2017
Total Direct Costs: $100,000

Role: Principal Investigator, Percent Effort: 5%; Co-Principal Investigator: NM King, PhD
Title: "Potential Anti-Tumorigenic Antigen for Cancer Therapy." To identify and test a target that cross-reacts with the CD11b antibody and determines its efficacy in killing both sensitive and chemoresistant ovarian cancer cells.
Source: Elsa U. Pardee Foundation Grant Program, Proposal #17-0715
Date: 01/01/18 – 12/31/18
Total Direct Costs: $187,958

Role: Principal Investigator, Percent Effort: NA
Title: "A Novel Target with Intriguing Anti-Tumorigenic Effects in Cancer." To identify and test a target that cross-reacts with the CD11b antibody and determines it efficacy in killing both sensitive and chemoresistant ovarian cancer cells.
Source: Ovarian Cancer Research Fund Alliance, Inc. (OCRFA)
Date: 01/01/18 – 12/31/20
Total Direct Costs: $300,000

Role: Mentor, Percent Effort: 0%; Principal Investigator: NM King, PhD
Title: "Antitumor Effects of Targeting Integrin αV/β1 in Ovarian Cancer Cells." To test the anti-tumorigenic potential of integrin αV/β1 antibodies in ovarian cancer patient samples.
Source: Ovarian Cancer Research Fund Alliance, Inc., Ann Schreiber Mentored Investigator Award
Date: 01/01/18 – 12/31/18

Total Direct Costs: $75,000

Pending Other Grants and Contracts

Role: Principal Investigator
Title: "Repurposing ABCIXIMAB, A Clinically Approved Anticoagulant for the Treatment of Ovarian Cancer." To determine whether abciximab is an effective therapy against sensitive and resistant ovarian cancer.
Source: Michigan Ovarian Cancer Alliance (MIOCA)
Date: 04/01/17 – 03/31/18
Total Direct Costs: $50,000

Role: Principal Investigator
Title: "ReoPro and Ovarian Cancer."
Source: Michigan Ovarian Cancer Alliance (MIOCA)
Date: 04/01/17 – 03/31/18
Total Direct Costs: $50,000

Role: Principal Investigator
Title: "A Novel Target with Intriguing Anti-Tumorigenic Effects in Cancer."
Source: DS Biotech, LLC, Proposal #17-0289
Date: 07/01/17– 06/30/18
Total Direct Costs: $100,000

Submitted National/International Grants and Contracts

Role: Principal Investigator
Title: "A Novel Target with Intriguing Anti-Tumorigenic Effects in Ovarian Cancer."
Source: Rivkin Center for Ovarian Cancer, Pilot Study Awards, 573569

Previously Funded Grants and Contracts

Role: Co-Principal Investigator; Principal Investigators: MP Diamond, MD, EN Kraiselburd, PhD
Title: "WSU-UPR Research Partnership to Promote Diversity in the Reproductive Sciences"
Source: NIH/NICHD, HD-09-008
Date: 08/2010 – 07/2015
Total Direct Costs: $3,020,000

Role: Co-Principal Investigator, Principal Investigator: MP Diamond, MD
Title: "WSU Clinical and Translational Science Award Planning Grant"
Source: NIH/NICHD, 1P20 RR 023578
Date: 09/2006 – 09/2012
Total Direct Costs: $2,225,750

Role: Consultant; Principal Investigator: MP Diamond, MD
Title: "WSU Cooperative Reproductive Medicine Network Center"
Source: NIH/NICHD, U10 HD-39005
Date: 08/2007 – 07/2012
Total Direct Costs: $1,510,000
Role: Principal Investigator, Percent Effort: 3.60%

Title: "Postoperative Adhesion: Roles of Hypoxia and Nitric Oxide"
Source: NIH/NICHD, Division of Pharmacology, Physiology, and Biological Chemistry, 1R01 GM069941-01A3
Date: 10/01/06 – 09/30/12
Total Direct Costs: $1,312,500

Role: Mentor; Principal Investigator: J White, MS, PhD Candidate (WSU)
Title: "Post-Operative Adhesions: Roles of Hypoxia in Nitric Oxide"
Source: NIH/NICHD, Minority Research Supplemental Award, 3R01GM069941-02S1
Date: 01/01/08 – 08/31/10
Total Direct Costs: $151,441

Role: *Principal Investigator
Title: "*CUAAH Subcontract – Specialty Laboratory Core"
Date: 06/01/08 – 05/31/10
Total Direct Costs: $403,840

Principal Investigator: JM Flack, MD
Title: "Center for Urban African American Health (CUAAH)"
Source: NIH/NIEHS
Date: 06/01/07 – 05/31/10
Total Direct Costs for Center: $9,487,709

Role: Principal Investigator
Title: "Angiogenesis of Ovarian Cancer"
Source: Frank Iacobell Endowed Chair, Department of Obstetrics and Gynecology, Wayne State University School of Medicine
Date: 01/01/08 – 12/31/09
Total Direct Costs: $41,500

Role: Co-Principal Investigator; Principal Investigator: R Kannan, PhD
Title: "Wayne State University, Department of Engineering – Subcontract"
Source: President's Research Award, Technology and Transfer Office
Date: 01/01/08 – 12/31/09
Total Direct Costs: $15,000

Role: Consultant; Principal Investigator: MP Diamond, MD U10 HD-39005
Title: "WSU Cooperative Reproductive Medicine Network Center"
Source: NIH/NICHD
Date: 04/01/00 – 03/31/07
Total Direct Costs: $1,349,994

Role: Principal Investigator; Co-Principal Investigator: MP Diamond, MD
Title: "Testing of Perfluorodecalin for Adhesion Prevention"
Source: Novel Pharma, Inc.
Date: 11/01/01 – 06/30/02
Total Direct Costs: $32,000

Role: Principal Investigator; Co-Principal Investigator: MP Diamond, MD
Title: "Effect of Tissel on Human Peritoneal Fibroblasts"
Source: Baxter Research Grant
Date: 09/30/01 – 12/31/02
Total Direct Costs: $98,000

Role: Co-Principal Investigator; Principal Investigator: MP Diamond, MD
Title: "Why Does Endometriosis Cause Adhesions?"
Source: Endometriosis Association
Date: 01/01/01 – 12/31/01
Total Direct Costs: $38,000

Role: Co-Principal Investigator; Principal Investigator: MP Diamond, MD
Title: "Effect of Tissel on Human Mesothelial Cell Culture"
Source: Baxter Research Grant
Date: 01/01/01 – 08/31/01
Total Direct Costs: $60,000

Role: Principal Investigator
Title: "The Effects of Hypoxia on the Levels of Peritoneal ECM Proteins"
Source: Wayne State University Department of Obstetrics and Gynecology, Interdepartmental Research Grant
Date: 03/01/98 – 12/31/00
Total Direct Costs: $19,000

Role: Principal Investigator
Title: "The Role of p53 in the Pathogenesis of Keloids"
Source: Henry Ford Hospital Small Project Award
Date: 01/01/98 – 12/31/98
Total Direct Costs: $20,000

Role: Principal Investigator
Title: "Patterns of Cytokine Expression in Cutaneous T-Cell Lymphoma"
Source: Henry Ford Hospital Small Project Award
Date: 01/01/93 – 12/31/94
Total Direct Costs: $20,000

Previously Submitted, Not Funded Grants and Contracts

Role: Principal Investigator, Percent Effort: 20%
Title: "Novel Mechanisms of Apoptosis in Chemoresistant Ovarian Cancer Cells." To determine whether chenoresistance in ovarian cancer manifests decreased apoptosis through enchanged s-nitrosylation of caspase-3 mechanism, which can be reversed by DCA.

Source: NIH/NICHD, R21
Date: 04/01/17 – 03/31/19
Total Direct Costs: $423,500

Role: Principal Investigator
Title: "Novel Mechanisms of Apoptosis in Chemoresistant Ovarian Cancer Cells." To determine whether chenoresistance in ovarian cancer manifests decreased apoptosis through enchanged s-nitrosylation of caspase-3 mechanism, which can be reversed by DCA.
Source: Elsa U. Pardee Foundation Grant Program
Date: 01/01/17 – 12/31/17
Total Direct Costs: $113,966

Role: Mentor, Percent Effort: 0%; Principal Investigator: NM King, PhD
Title: "Novel Mechanisms of Apoptosis in Chemoresistant Ovarian Cancer Cells." The project's design is to determine whether chemoresistance in ovarian cancer manifests decreased apoptosis through enhanced s-nitrosylation of caspase-3 mechanism, which can be reversed by DCA.
Source: NIH/NICHD, R03
Date: 12/01/16 – 11/30/18
Total Direct Costs: $50,000

Role: Principal Investigator
Title: "Innovative New Target for Ovarian Cancer Therapy." The project was designed to identify and test a target that cross-reacts with the CD11b antibody and determines its efficacy in killing both sensitive and chemoresistant ovarian cancer cells.
Source: The Honorable Tina Brozman Foundation, Inc. for Ovarian Cancer Research
Date: 08/01/16 – 07/31/18
Total Direct Costs: $200,000

Role: Principal Investigator
Title: "Redox Enzyme-Mediated Prosurvival of Chemoresistance in Ovarian Cancer." To determine whether development of chenoresistance in ovarian cancer is attributed to enhanced oxidative stress leading to a genotype switch in key oxidant and antioxidant enzymes.
Source: Ovarian Cancer Research Fund Alliance, Inc. (OCRFA)
Date: 2016 – 2019
Total Direct Costs: $900,000

Role: Mentor, Percent Effort: 0%; Principal Investigator: NM King, PhD
Title: "Chemoresistant Ovarian Cancer Cells Manifest Lower Vascular Endothelial Growth Factor and Hypoxia Inducible Factor-1α: A Potential Survival Mechanism." The design of the project was to determine whether VEGF and HIF-1α contribute to the persistence of chemoresistance in ovarian cancer.
Source: Ovarian Cancer Research Fund, Ann Schreiber Mentored Investigator Award
Date: 2016 – 2017
Total Direct Costs: $75,000

Role: Mentor, Percent Effort: 0%; PI: NM King, PhD
Title: "Novel Biomarkers for Early Detection of Ovarian Cancer." The design of the project was to determine whether MPO and free iron could be utilized as biomarkers for the early detection of ovarian cancer.
Source: NIH/NICHD, R03 – Resubmission of scored proposal

Date: 12/01/15–11/30/17
Total Direct Costs: $153,583

Role: Principal Investigator, Percent Effort: 30%
Title: "Redox Enzyme-Mediated Prosurvival of Chemoresistance in Ovarian Cancers." The design of the project was to determine whether development of chemoresistance in ovarian cancer is attributed to enhanced oxidative stress leading to a genotype switch in key oxidant and antioxidant enzymes.
Source: NIH/NICHD, R01
Date: 12/01/15 – 11/30/20
Total Direct Costs: $2,494,526

Role: Mentor, Percent Effort: 0%; PI: NM King, PhD
Title: "Novel Biomarkers for Early Detection of Ovarian Cancer." The design of the project was to determine whether MPO and free iron could be utilized as biomarkers for the early detection of ovarian cancer.
Source: NIH/NICHD, R03
Date: 11/01/14 – 10/31/16
Total Direct Costs: $152,000

Role: Principal Investigator, Percent Effort: 30%
Title: "Chemoresistance Induces a Genotype Switch in Redox Enzymes in Ovarian Cancer." The design of the project was to determine whether development of chemoresistance in ovarian cancer is attributed to enhanced oxidative stress leading to a genotype switch in key oxidant and antioxidant enzymes.
Source: NIH/NICHD, R01
Date: 04/ 01/15 – 03/31/20
Total Direct Costs: $3,124,495

Role: Mentor, Percent Effort: 0%; Principal Investigator: NM King, PhD
Title: "Biomarkers for Early Detection of Ovarian Cancer." The design of the project was to determine whether MPO and free iron could be utilized as biomarkers for the early detection of ovarian cancer.
Source: Sandy Rollman Ovarian Cancer Foundation (SROCF) Fellowship
Date: 06/01/14 – 05/31/15
Total Direct Costs: $50,000

Role: Mentor, Percent Effort: 0%; Principal Investigator: NM King, PhD
Title: "Novel Biomarkers for Early Detection of Ovarian Cancer." The design of the project was to determine whether MPO and free iron could be utilized as biomarkers for the early detection of ovarian cancer.
Source: Ladies Auxiliary to the Veterans of Foreign Wars, Postdoctoral Cancer Research Fellowship
Date: 06/01/14 – 05/31/16
Total Direct Costs: $50,000

Role: Mentor, Percent Effort: 0%; PI: NM King, PhD
Title: "Novel Biomarkers for Early Detection of Ovarian Cancer." The design of the project was to determine whether MPO and free iron could be utilized as biomarkers for the early detection of ovarian cancer.
Source: Kaleidoscope of Hope Foundation, Young Investigator Award

Date: 04/01/14 – 03/31/15
Total Direct Costs: $50,000

Role: Mentor, Percent Effort: 0%; Principal Investigator: NM King, PhD
Title: "Novel Biomarkers for Early Detection of Ovarian Cancer." The design of the project was to determine whether MPO and free iron could be utilized as biomarkers for the early detection of ovarian cancer.
Source: Damon Runyon Cancer Research Foundation
Date: 07/01/14 – 06/30/17
Total Direct Costs: $158,000

Role: Mentor, Percent Effort: 0%; Principal Investigator: NM King, PhD
Title: "Chemoresistance Induces a Genotype Switch in Epithelial Ovarian Cancer Cells." The project was designed to determine whether development of chemoresistance in ovarian is attributed to enhanced oxidative stress leading to a genotype switch in key oxidant and antioxidant enzymes.
Source: American Cancer Society Postdoctoral Fellowship
Date: 01/01/15 – 12/31/18
Total Direct Costs: $163,500

Role: Mentor, Percent Effort: 0%; Principal Investigator: NM King, PhD
Title: "Novel Biomarkers for the Early Detection of Ovarian Cancer." The design of the project was to determine whether MPO and free iron can be utilized as biomarkers for the early detection of ovarian cancer.
Source: American Association for Cancer Research
Date: 2015 – 2016
Total Direct Costs: $50,000

Role: Principal Investigator, Percent Effort: 30%
Title: "Postoperative Adhesion Development is Controlled by Mechanisms that Emanate from a Hypoxia-Induced Genotype Switch in Key Enzymes of Oxidative Stress." Identification of markers that are strongly associated with adhesions and in patients will contribute to both the delineation of mechanisms of adhesion development and serve as potential targets for therapy and intervention.
Source: NIH/NICHD, R01
Date: 07/01/15 – 06/30/20
Total Direct Costs: $1,921,633

Role: Principal Investigator, Percent Effort: 25%
Title: "Combination of Antioxidants Effectively Reduces Adhesion Development." The design of the project was to determine the effects of antioxidants on the prevention of postoperative adhesion development.
Source: NIH/NICHD, R03
Date: 07/01/15 – 06/30/17
Total Direct Costs: $153,314

Role: Mentor, Percent Effort: 0%; Principal Investigator: NM King, PhD
Title: "New Insights into the Pathogenesis of Ovarian Cancer." To identify keymarkers of oxidative stress that have the potential to serve as screening tools for ovarian cancer and may play a role in the acquisition of chemoresistance.
Source: Prevent Cancer Foundation

Date: 04/01/14 – 01/31/16
Total Direct Costs: $80,000

Role: Mentor, Percent Effort: 0%; Principal Investigator: AO Awonuga, MD
Title: "Effects of Dietary Lycopene on Incidence and Severity of Postoperative Adhesions." The design of the project was to determine the effects of antioxidants on the prevention of postoperative adhesion development.
Source: NIH/NICHD, R03
Date: 09/01/14 – 08/31/16
Total Direct Costs: $152,000

Role: Mentor, Percent Effort: 0%; Principal Investigator: NM King, PhD
Title: "Novel Biomarkers for Detection of Early Ovarian Cancer." The design of the project was to determine whether MPO and free iron can be utilized as biomarkers for the early detection of ovarian cancer.
Source: Marsha Rivkin Center for Ovarian Cancer Research, Scientific Scholar Award – Postdoctoral Fellowship
Date: 04/01/14 – 03/31/15
Total Direct Costs: $60,000

Role: Mentor; Percent Effort: 0%; Principal Investigator: J Belotte, MD
Title: "Catalase SNP as a Genetic Predictor for Epithelial Ovarian Cancer." The design of the project was to determine whether a SNP in the catalase gene can be utilized as a predictive marker for epithelial ovarian cancer.
Source: Marsha Rivkin Center for Ovarian Cancer Research, Scientific Scholar Award – Postdoctoral Fellowship
Date: 04/01/14 – 03/31/15
Total Direct Costs: $60,000

Role: Mentor, Percent Effort: 0%; Principal Investigator: NM King, PhD
Title: "Novel Biomarkers for the Early Detection of Ovarian Cancer." The design of the project was to determine whether MPO and free iron can be utilized as biomarkers for the early detection of ovarian cancer.
Source: Hope Funds Cancer Research Postdoctoral Fellowship
Date: 2014 – 2016
Total Direct Costs: $100,000

Role: Principal Investigator
Title: "Chemoresistance in Ovarian Cancer Manifests a Genotype Switch in Oxidant Enzymes."
The design of the project was designed to determine whether a genotype switch in key oxidant enzymes is induced in chemotherapy treated ovarian cancer cells and the subsequent effect of the enzymatic activity.
Source: Marsha Rivkin Center for Ovarian Cancer Research; Pilot Study
Date: 04/01/14 – 03/31/15
Total Direct Costs: $75,000

Role: Principal Investigator, Percent Effort: 30%; Co-Investigators: MP Diamond, MD, S Ghamande, PhD
Title: "Chemoresistance Induces a Genotype Switch in Redox Enzymes in Ovarian Cancer."
The design of the project was to determine whether development of chemoresistance in ovarian

cancer were attributed to enhanced oxidative stress leading to a genotype switch in key oxidant and antioxidant enzymes.
Source: NIH/NICHD, R01
Date: 07/01/14 – 06/30/19
Total Direct Costs: $2,932,687

Role: Principal Investigator, Percent Effort: 25%
Title: "Chemoresistance in Ovarian Cancer is Attributed to Enhanced Oxidative Stress." The design of the project was to determine whether development of chemoresistance in ovarian cancer were attributed to enhanced oxidative stress.
Source: NIH/NICHD, R03
Date: 07/01/14 – 06/30/16
Total Direct Costs: $152,000

Role: Principal Investigator, Percent Effort: 25%
Title: "Chemoresistance in Ovarian Cancer Manifests a Genotype Switch in Oxidant Enzymes." The design of the project was to determine whether development of chemoresistance in ovarian cancer were attributed to enhanced oxidative stress leading to a genotype switch in key oxidant and antioxidant enzymes.
Source: NIH/NICHD, R03
Date: 07/01/14 – 06/30/16
Total Direct Costs: $152,000

Role: Mentor, Percent Effort: 0%; Principal Investigator: J Belotte, MD
Title: "Characterization of Epithelial Ovarian Cancer Stem Cells." The design of the project was to determine the role of pluripotency markers in epithelial ovarian cancer and the association with survival.
Source: NIH/NICHD, R03
Date: 09/30/14 – 06/30/16
Total Direct Costs: $152,000

Role: Mentor, Percent Effort: 0%; PI: J Belotte, MD
Title: "Catalase SNP as a Genetic Predictor for Epithelial Ovarian Cancer." The design of the project was to determine whether a SNP in the catalase gene can be utilized as a predictive marker for epithelial ovarian cancer.
Source: NIH/NICHD, R03
Date: 09/30/14 – 08/31/16
Total Direct Costs: $152,000

Role: Principal Investigator, Percent Effort: 20%
Title: "Innovative New Target for Ovarian Cancer Therapy." The project was designed to identify and test a target that cross-reacts with the CD11b antibody and determines its efficacy in killing both sensitive and chemoresistant ovarian cancer cells.
Source: NIH/NICHD, R21
Date: 12/31/16 – 11/30/18
Total Direct Costs: $275,000

Role: Principal Investigator
Title: "Redox Enzyme-Mediated Prosurvival of Chemoresistance in Ovarian Cancer." The design of the project was to determine whether development of chemoresistance in ovarian

cancer were attributed to enhanced oxidative stress leading to a genotype switch in key oxidant and antioxidant enzymes.
Source: Ovarian Cancer Research Fund, Program Project Development Grant
Date: 201 6 – 2019
Total Direct Costs: $900,000

Role: Principal Investigator, Percent Effort: 20%
Title: "Novel Mechanisms of Apoptosis in Chemoresistant Ovarian Cancer Cells." The design of the project was to determine whether chemoresistance in ovarian cancer manifests decreased apoptosis through enhanced s-nitrosylation of caspase-3 mechanism, which can be reversed by DCA.
Source: NIH/NICHD, R21
Date: 12/01/16 – 11/30/18
Total Direct Costs: $275,000

Role: Mentor, Percent Effort 0%; Principal Investigator: NM King, PhD
Title: "Novel Biomarkers for Early Detection of Ovarian Cancer." The design of the project was to determine whether MPO and free iron can be utilized as biomarkers for the early detection of ovarian cancer.
Source: L'Oreal USA for Women in Science Fellowship
Date: 2016 – 2017
Total Direct Costs: $60,000

PATENTS:

Status:	Provisional Submission
Date:	2016
Number:	WSU
Title:	Compositions and Methods Targeting CD11b/CD18, Myeloperoxidase and/or Integrin Alpha and Beta 1 to Treat Solid Tumors
Role:	Inventor

Status:	Pending
Date:	2015
Number:	WSU
Title:	Novel Approach to Selectively Kill Cancer Cells
Role:	Inventor

Status:	Pending
Date:	2008
Number:	WSU
Title:	Anticancer Vaccine
Role:	Inventor

Status: Pending
Date: WSU
Number: WSU 00-492
Title: Prevention of Adhesions
Role: Inventor

Status: Pending
Date: March 18, 2004
Number: WSU
Title: Regulation of Peritoneal Healing and Adhesion Development
Role: Inventor

Status: Pending
Date: June 25, 2002
Number: WSU
Title: Modification of Healing and Adhesion Development
Role: Inventor

PUBLICATIONS

Peer-Reviewed Publications

**Indicates student, trainee, or postdoctoral*

Reports of Original Work

1. **Saed GM**, *Fletcher NM, Diamond MP, Morris RT, Gomez-Lopez N, *Memaj I. Novel expression of CD11b in epithelial ovarian cancer: potential therapeutic target. Gynecologic Oncology 2018 Mar;148(3):567-575. [Epub 2018 Jan 10] PMID: 29329880. *Role: Writer and Mentor*

2. Detti L, *Fletcher NM, **Saed GM**, Peregrin-Alvarez I, Uhlmann RA. Anti-Műllerian Hormone (AMH) may stall ovarian cortex function through modulation of hormone receptors other than the AMH receptor. Reproductive Sciences January 1:19337119117737850, 2017. [Epub ahead of print] PMID: 29141508. *Role: Mentor and collaborator*

3. *Fletcher NM, *Abusamaan MS, *Memaj I, *Saed MG, Al-Hendy A, Diamond MP, **Saed GM**. Oxidative stress: a key regulator of leiomyoma cell survival. Fertility and Sterility 107(6):1387-1394.e1 , 2017. Epub May 5, 2017. PMID: 28483502. *Role: Writer and Mentor*

4. **Saed GM**, Diamond MP, Fletcher NM. Updates of the role of oxidative stress in the pathogenesis of ovarian cancer. Gynecologic Oncology 2017 Jun;145(3):595-602. Epub 2017 Feb 23. Review. PMID:28237618. *Role: Writer and Mentor*

5. *Fletcher NM, *Belotte J, *Saed MG, *Memaj I, Diamond MP, Morris RT, **Saed GM**. Specific point mutations in key redox enzymes are associated with chemoresistance in epithelial ovarian cancer. Free Radical Biology and Medicine 102:122-132, 2017. Epub November 25, 2016. PMID: 27890641. *Role: Mentor and Writer*

6. *Nusrat O, *Belotte J, *Fletcher NM, *Memaj I, *Saed MG, Diamond MP, **Saed GM**. The role of angiogenesis in the persistence of chemoresistance in epithelial ovarian cancer. Reproductive Sciences 23(11):1484-1492, 2016. Epub April 26, 2016. PMID: 27122375 *Role: Mentor and Writer*

7. *Shaeib F, *Khan SN, *Thakur M, Kohan-Ghadr HR, Drewlo S, **Saed GM**, Pennathur S, Abu-Soud HM. The impact of myeloperoxidase and activated macrophages on metaphase II mouse oocyte quality. PLoS One 11(3):e0151160, 2016. eCollection 2016. PMID: 26982351 *Role: Mentor and collaborator*

8. Ahmed RS, Liu G, Renzetti A, Farshi P, Yang H, Soave C, **Saed G**, El-Ghoneimy AA, El-Banna HA, Foldes R, Chan TH, Dou QP. Biological and Mechanistic Characterization of Novel Prodrugs of Green Tea Polyphenol Epigallocatechin Gallate Analogs in Human Leiomyoma Cell Lines. J Cell Biochem. 2016 Oct;117(10):2357-69.doi: 10.1002/jcb.25533. Epub 2016 Mar 28. PMID:26950525. *Role: Collaborator*

9. *Fletcher NM, Awonuga AO, *Abusamaan MS, *Saed MG, Diamond MP, **Saed GM**. Adhesion phenotype manifests an altered metabolic profile favoring glycolysis. Fertility and Sterility 105(6):1628-1637, 2016. Epub February 23, 2016. PMID: 26920255. *Role: Mentor and Writer.*

10. **Saed GM**, *Fletcher NM, Diamond MP. The creation of a model for ex vivo development of postoperative adhesions. Reproductive Sciences 23(5):610-622, 2016. Epub September 25, 2015. PMID: 26408397. *Role: Mentor*.

11. *Belotte J, *Fletcher NM, *Saed MG, *Abusamaan MS, *Dyson G, Diamond MP, **Saed GM**. A single nucleotide polymorphism in catalase is strongly associated with ovarian cancer survival. PLoS One 10(8):e0135739, 2015. eCollection 2015. PMID: 26301412 PMCID: PMC4547699. *Role: Mentor and Writer*

12. *Fletcher NM, Awonuga AO, *Neubauer BR, *Abusamaan MS, *Saed MG, Diamond MP, **Saed GM**. Shifting anaerobic to aerobic metabolism stimulates apoptosis through modulation of redox balance: potential intervention in the pathogenesis of postoperative adhesions. Fertility and Sterility 104(4):1022-1029, 2015. Epub July 26, 2015. PMID: 26215756. *Role: Mentor and Writer*

13. *Khan SN, *Shaeib F, *Najafi T, *Kavdia M, Gonik B, **Saed GM**, Goud PT, Abu-Soud HM. Diffused intra-oocyte hydrogen peroxide activates myeloperoxidase and deteriorates oocyte quality. PLoS One 10(7):e0132388, 2015. eCollection 2015. PMID: 26197395 PMCID: PMC4511228 *Role: Mentor, collaborator, assisted with experimental format*

14. *****Fortin CN, **Saed GM**, Diamond MP. Predisposing factors to post-operative adhesion development. Hum Reprod Update. 2015 Jul-Aug;21(4):536-51.doi: 10.1093/humupd/dmv021. Epub 2015 May 1. Review. PMID:25935859. *Role: Mentor and Writer*

15. *Shaeib F, *Khan SN, *Ali I, *Najafi T, *Maitra D, *Abdulhamid I, **Saed GM**, Pennathur S, Abu-Soud HM. Melatonin prevents myeloperoxidase heme destruction and the generation of free iron mediated by self-generated hypochlorous acid. PLoS One 10(3):e0120737, 2015. eCollection 2015. PMID: 25835505 PMCID: PMC4383586 *Role: Mentor, collaborator, assisted with writing of article*

16. *Maitra D, *Ali I, *Abdulridha RM, *Shaeib F, *Khan SN, **Saed GM**, Pennathur S, Abu-Soud HM. Kinetic studies on the reaction between dicyanocobinamide and hypochlorous acid. PLoS One 9(11):e110595, 2014.PMID: 25375773. PMCID: PMC4222763. *Role: Mentor, collaborator, assisted with manuscript writing*

17. Abu-Soud HM, *Maitra D, *Shaeib F, *Khan SN, *Byun J, *Abdulhamid I, *Yang Z, **Saed GM**, Diamond MP, Andreana PR, Pennathur S. Disruption of heme-peptide covalent cross-linking in mammalian peroxidases by hypochlorous acid. Journal of Inorganic Biochemistry 140:245-254, 2014. Epub July 8, 2014. PMID: 25193127 PMCID: PMC4449957. *Role: Mentor and collaborator*.

18. *Belotte J, *Fletcher NM, *Alexis M, Morris RT, Munkarah AR, Diamond MP, **Saed GM**. Sox2 gene amplification significantly impacts overall survival in serous epithelial ovarian cancer. Reproductive Sciences 22(1):38-46, 2015. Epub July 18, 2014. PMID: 25038052. PMCID: PMC4275450. *Role: Mentor and Writer*

19. Goud PT, Goud AP, *Najafi T, Gonik B, Diamond MP, **Saed GM**, Zhang X, Abu-Soud HM. Direct real-time measurement of intra-oocyte nitric oxide concentration in vivo. PLoS One 9(6):e98720, 2014. PMID: 24887331 PMCID: PMC4041775 *Role: Collaborator and assisted with manuscript writing.*

20. *Awonuga AO, *Belotte J, *Abuanzeh S, *Fletcher NM, Diamond MP, **Saed GM**. Advances in the Pathogenesis of Adhesion Development: The Role of Oxidative Stress. Reprod Sci. 2014 Jul;21(7):823-836. Epub 2014 Feb 11. Review. PMID:24520085. Role: Mentor and Writer.

21. *Fletcher NM, *Saed MG, *Abuanzeh S, Abu-Soud HM, Al-Hendy A, Diamond MP, **Saed GM**. Nicotinamide adenine dinucleotide phosphate oxidase is differentially regulated in normal myometrium versus leiomyoma. Reproductive Sciences 21(9):1145-1152, 2014. Epub February 11, 2014. PMID: 24520084 *Role: Mentor and Writer.*

22. *Fletcher NM, *Abuanzeh S, *Saed MG, Diamond MP, Abu-Soud HM, **Saed GM**. Nicotinamide adenine dinucleotide phosphate oxidase expression is differently regulated to favor a pro-oxidant state that contributes to postoperative adhesion development. Reproductive Sciences 21(8):1050-1059, 2014. Epub February 10, 2014. PMID: 24516041. *Role: Mentor and Writer*

23. *Detti L, *Uhlmann RA, *Zhang J, Diamond MP, **Saed GM**, *Fletcher NM, Lu M, Williams LJ. Goserelin fosters bone elongation, but does not prevent ovarian damage in cyclophosphamide-treated prepubertal mice. Fertility and Sterility 101(4):1157-1164.e.1, 2014. Epub January 23, 2014. PMID: 24462062. *Role: Collaborator and mentor*

24. *Fletcher NM, Awonuga AO, *Saed MG, Abu-Soud HM, Diamond MP, **Saed GM**. Lycopene, a powerful antioxidant, significantly reduces the development of the adhesion phenotype. Systems Biology in Reproductive Medicine 60(1):14-20, 2014. Epub November 12, 2013. PMID: 24219141. *Role: Mentor and Writer*

25. *†Belotte J, *†Fletcher NM, Awonuga AO, *Alexis M, Abu-Soud HM, Saed MG, Diamond MP, **Saed GM**. The role of oxidative stress in the development of Cisplatin resistance in epithelial ovarian cancer. †Denotes co-authorship. Reproductive Sciences 21(4):503-508, 2014. Epub September 27, 2013. PMID: 24077440 PMCID: PMC3960837

26. Detti L,* Uhlmann RA, Lu M, Diamond MP, **Saed GM**, *Fletcher NM, Zhang J, Williams LJ. Serum markers of ovarian reserve and ovarian histology in adult mice treated with cyclophosphamide in pre-pubertal age. Journal of Assisted Reproduction and Genetics 30(11):1421-1429, 2013. Epub September 24, 2013. PMID: 24057193, PMCID: PMC3879939*. Role: Collaborator and mentor, assisted with experimental design.*

27. Detti L, Uhlmann RA, *Fletcher NM, Diamond MP, **Saed GM**. Endometrial signaling pathways during ovarian stimulation for assisted reproduction technology. Fertility and Sterility 100(3): 889-894, 2013. Epub June 24, 2013. PMID: 23806847 PMCID: PMC3880797*.*

28. *Banerjee J, *Shaeib F, *Maitra D, **Saed GM**, Dai J, Diamond MP, Abu-Soud HM. Peroxynitrite affects the cumulus cell defense of metaphase II mouse oocytes leading to disruption of the spindle structure in vitro. Fertility and Sterility 100(2):578-584.e1, 2013. Epub May 28, 2013. PMID: 23721714*. Role: Mentor and collaborator*

29. *Maitra D, *Shaeib F, *Abdulhamid I, *Abdulridha RM, **Saed GM**, Diamond MP, Pennathur S, Abu-Soud HM. Myeloperoxidase acts as a source of free iron during steady-state catalysis by a feedback inhibitory pathway. Free Radical and Biological Medicine 63:90-98, 2013. Epub April 25, 2013. PMID: 23624305 PMCID:PMC3863623. *Role: Mentor and collaborator*

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Non Peer-Reviewed Publications

Other (On-Line Publications) * Indicates student, trainee, or postdoctoral

1. *Nusrat O, *Belotte J, *Fletcher NM, *Memaj I, *Saed MG, Diamond MP, **Saed GM**. The role of angiogenesis in the persistence of chemoresistance in epithelial ovarian cancer. www.OncToday.com, Beyond the Abstract, June 21, 2016.

2. *Belotte J, *Fletcher NM, *Alexis M, Morris RT, Munkarah AR, Diamond MP, **Saed GM**. Sox2 gene amplification significantly impacts overall survival in serous epithelial ovarian cancer. Global Medical Discovery Series (*Key Scientific Article Contributing to Excellence in Biomedical Research*), summer issue 2015.

3. *Fletcher NM, *Saed MG, Abu-Soud HM, Al-Hendy A, Diamond MP, **Saed GM**. Uterine fibroids are characterized by an impaired antioxidant cellular system: potential role of hypoxia in the pathophysiology of uterine fibroids. Featured article. MDLinx.com/ obstetrics-gynecology/ news-article, November 2013.

PRESENTATIONS

Podium Presentations (Referred)

1. *Novel Target for Ovarian Cancer Immunotherapy*. 48th Annual Meeting of the Society of Gynecologic Oncology's Women's Cancer, National Harbor, MD, March 2017.

2. *Targeting Integrin αV/β1 Receptor Manifests Intriguing Anti-Tumor Effects in Sensitive and Chemoresistant Ovarian Cancer Cells: Potential Therapeutic Target*. 64th Annual Scientific Meeting of the Society for Reproductive Investigation, Orlando, FL, March 2017.

3. *Human Adhesion Fibroblasts are Characterized by Reduction in the level* of *Pluripotency Markers as Compared to Normal Peritoneal Fibroblasts*. 72nd Annual Meeting of the American Society for Reproductive Medicine, Salt Lake City, UT, October 2016.

4. *Anti-Mullerian Hormone (AMH) for Prevention of Tissue Activation after Vitrified/Thawed Ovarian Cortex Xenotransplantation*. 72nd Annual Meeting of the American Society for Reproductive Medicine, Salt Lake City, UT, October 2016.

5. *Dichloroacetate Induces Apoptosis of Uterine Leiomyoma Cells Through A Mechanism Involving Modulation of Oxidative Stress*. 63rd Annual Meeting of the Society for Reproductive Investigation, Montreal, Quebec, Canada, March 2016.

6. *Chemoresistance in Epithelial Ovarian Cancer Cells is Controlled by Mechanisms Emanating from Chemotherapy-Induced Genotype Switch in Glutathione Peroxidase, Through the Up-Regulation of Cytidine Deminase*. 62nd Annual Meeting of the Society for Reproductive Investigation, San Francisco, CA, March 2015.

7. *Elevated Serum Anti-Müllerian Hormone (AMH) Stalls Ovarian Follicle Development by Downregulating FSH- and LH-Receptors and Inhibin-B Production*. Proceedings of the 71st Annual Meeting of the American Society for Reproductive Medicine, Baltimore, MD, October 2015.

8. *Hypochlorous Acid Reversibly Inhibits Caspase-3: A Potential Regulator of Apoptosis*. Joint Meeting of the 22nd Society for Redox Biology and Medicine (SFRBM) and 17th Society for Free Radical Research International (SFRRI), Boston, MA, November 2015.

9. *The In-Vivo Effects of Superoxide Dismutase on the Incidence and Severity of Post-Operative Adhesion Development*. 70th Annual Meeting of the American Society for Reproductive Medicine, Honolulu, HI, October 2014.

10. *Superoxide Dismutase Significantly Delayed the Development of Cisplatin Resistance in Epithelial Ovarian Cancer Cells*. American Association for Cancer Research's Precision Medicine Series: Drug Sensitivity and Resistance. Improving Cancer Therapy Special Conference, Orlando, FL, June 2014.

11. *Chemoresistant Ovarian Cancer Cells Manifest Lower Vascular Endothelial Growth Factor and Hypoxia Induced Factor-1α: A Potential Survival Mechanism*. American Association for Cancer Research's Precision Medicine Series: Drug Sensitivity and Resistance. Improving Cancer Therapy Special Conference, Orlando, FL, June 2014.

12. *Dicholoroacetate Increases Sensitivity to Chemotherapy by Modulation of Antioxidants in Epithelial Ovarian Cancer*. 61st Annual Meeting of the Society for Gynecologic Investigation, Florence, Italy, March 2014.

13. *Catalase and NADPH Oxidase Single Nucleotide Polymorphisms Are Associated with Increased Risk and Serve As Potential Targets for Breast and Ovarian Cancers*. 104th Annual Meeting of the American Association for Cancer Research, Washington, DC, April 2013.

14. *The Role of Oxidative Stress in the Development of Cisplatin Resistance in Epithelial Ovarian Cancer*. Poster session B. Advances in Ovarian Cancer Research: From Concept to Clinic. American Association for Cancer Research, Miami, FL, September 2013.

15. *Catalase and NADPH Oxidase Single Nucleotide Polymorphisms Are Associated with Increased Risk and Serve As Potential Targets for Breast and Ovarian Cancers*. 104th Annual Meeting of the American Association for Cancer Research, Washington, DC, April 2013.

16. *Endometrial Insulin Pathway during Ovarian Stimulation for Assisted Reproductive Technology (ART)*. 68th Annual Meeting of the American Society for Reproductive Medicine, San Diego, CA, October 2012.

17. *NADPH Oxidase p22-Phox Gene Polymorphism in Women is Associated with the Development of Postoperative Adhesions*. 59th Annual Meeting of the Society for Gynecologic Investigation, San Diego, CA, March 2012.

18. *Metabolism and Oxidative Stress: Integral Role in Regulation of the Adhesion Phenotype*. 58th Annual Meeting of the Society for Gynecologic Investigation, Miami Beach, FL, March 2011.

19. *Mass Spectrometric Identification of HOCl-Mediated Heme Degradation Products of Hemoglobin*. 59th ASMS Conference on Mass Spectrometry, Denver, CO, 2011.

20. *Inhibition of NADPH Oxidative Reductase Promotes Apoptosis in Epithelial Ovarian Cancer Cells*. 39th Annual Meeting of the Global Congress of Minimally Invasive Gynecology AAGL, Las Vegas, NV, November 2010.

21. *Reaction of Hemoglobin and Red Blood Cells with Hypochlorous Acid and Mechanism of Heme Destruction and Free Iron Release*. 17th Annual Meeting of the Society for Free Radical Biology and Medicine, Orlando, FL, November 2010.

22. *Liquid Chromatography Atmospheric Pressure Ionization Tandem: Mass Spectrometry Identifies Novel Hypochlorous Acid Reaction Products of Lycopene*. 58th Annual Meeting of the American Society of Mass Spectrometry, Salt Lake City, UT, May 2010.

23. *Role of Polychlorinated Biphenyls Enhancement of Lipid Peroxidation in Human Normal Peritoneal and Adhesion Fibroblasts*. 38th Annual Meeting of Global Congress of Minimally Invasive Gynecology AAGL, Orlando, FL, November 2009.

24. *Hydrogen Peroxide Bioavailability Determines the Sensitivity of Human Normal Peritoneal and Adhesion Fibroblasts to Hypoxia-Induced Lipid Peroxidation*. 38th Annual Meeting of Global Congress of Minimally Invasive Gynecology AAGL, Orlando, FL, November 2009.

25. *S-Nitrosylation of Caspase-3 Is the Mechanism by Which Adhesion Fibroblasts Manifest Lower Apoptosis*. 36th Annual Meeting of the American Association of Gynecologic Laparoscopists, Global Congress of Minimally Invasive Gynecology, Washington, DC, November 2007.

26. *Generation of Superoxide by Inducible Nitric Oxide Synthase in L-Arginine Deficient Fibroblasts Established From Human Adhesion Tissues*. 36th AAGL Annual Meeting, Global Congress of Minimally Invasive Gynecology, Washington, DC, November 2007.

27. *Hypoxia Stimulation of Expression of Type I Collagen and Fibronectin in Human Peritoneal and Adhesion Fibroblasts: Blockage by Interferon Gamma*. 36th AAGL Annual Meeting, Global Congress of Minimally Invasive Gynecology, Washington, DC, November 2007.

28. *Superoxide Induces the Adhesion Phenotype: Role of Hypoxia in the Pathogenesis of the Adhesion Development*. Global Congress of Minimally Invasive Gynecology, 36th Annual Meeting of the American Association of Gynecologic Laparoscopists, Washington, DC, November 2007.

29. *Nitric Oxide Synthase Isoforms are Differentially Expressed in Fibroblasts Isolated from Human Normal Peritoneum and Adhesion Tissues*. 63rd Annual Meeting of the American Society for Reproductive Medicine, Washington, DC, October 2007.

30. *Regulation of the Expression of INOS, COX-2, and VEGF in Postoperative Adhesions*. 62nd Annual Meeting of the American Society for Reproductive Medicine, New Orleans, LA, October 2006.

31. *Omega-3 Fatty Acid Prevents and Mitigates the Adhesion Phenotype in Normal Human Peritoneal and Adhesion Fibroblasts*. 62nd Annual Meeting of the American Society for Reproductive Medicine, New Orleans, LA, October 2006.

32. *IL6 Expression in Human Normal Peritoneal and Adhesion Fibroblasts: Regulation by Hypoxia*. 62nd Annual Meeting of the American Society for Reproductive Medicine, New Orleans, LA, October 2006.

33. *The Cross-Talk between Myeloperoxidase and Inducible Nitric Oxide Synthase in Post-operative Adhesions*. 62nd Annual Meeting of the American Society for Reproductive Medicine, New Orleans, LA, October 2006.

34. *TNF-Alpha Expression in Human Normal Peritoneal and Adhesion Fibroblasts: Regulation by Hypoxia*. 62nd Annual Meeting of the American Society for Reproductive Medicine, New Orleans, LA, October 2006.

35. *L-Arginine Deficiency in Fibroblasts Established from Human Adhesion Tissues Leads to the Generation of Superoxide by Inducible Nitric Oxide Synthase*. 53rd Annual Meeting of the Society for Gynecologic Investigation, Toronto, Ontario, Canada, March 2006.

36. *Regulation of Inducible Nitric Oxide Synthase in Post-Operative Adhesions*. 34th Annual Meeting of the American Association of Gynecologic Laparoscopists, Chicago, IL, November 2005.

37. *Cyclooxygenase-2 Inhibitors Enhance Apoptosis of Adhesion Fibroblasts*. 34th Annual Meeting of the American Association of Gynecologic Laparoscopists, Chicago, IL, November 2005.

38. *The Effects of Estradiol on the Expression of Estrogen, Progesterone, Androgen, and Prolactin Receptors in Human Peritoneal Fibroblasts*. 61st Annual Meeting of the American Society for Reproductive Medicine and the 1st Annual Meeting of the Canadian Fertility and Andrology Society, Palais des Congres, Montreal, Quebec, Canada, October 2005.

39. *Possible Role of Natural Immune Response against Fibroblasts in the Development of Post-Operative Adhesions*. 61st Annual Meeting of the American Society for Reproductive Medicine and the 51st Annual Meeting of the Canadian Fertility and Andrology Society, Palais des Congres, Montreal, Quebec, Canada, October 2005.

40. *Knockout of Inducible Nitric Oxide Expression Significantly Reduces the Expression of Type I Collagen and Transforming Growth Factor-β1 in Human Peritoneal and Adhesion Fibroblasts*. 61st Annual Meeting of the American Society for Reproductive Medicine and the 51st Annual Meeting of the Canadian Fertility and Andrology Society, Palais des Congres, Montreal, Quebec, Canada, October 2005. **Prize Paper Candidate**

41. *Regulation of Inducible Nitric Oxide Synthase in Post-Operative Adhesions*. 52nd Annual Meeting of the Society for Gynecologic Investigation, Los Angeles, CA, March 2005.

42. *Differential Expression of Myeloperoxidase (MPO) in Fibroblasts Isolated from Normal Peritoneal and Adhesion Tissues*. 4th International Peroxidase Meeting Joint with the 10th Myeloperoxidase Meeting, Shimokyo-Ku, Kyoto City, Japan, October 2004.

43. *Fibroblasts Isolated from Normal Peritoneal and Adhesion Tissues Differentially Express Myeloperoxidase (MPO)*. 60th Annual Meeting of the American Society for Reproductive Medicine, Philadelphia, PA, October 2004.

44. *Hypoxia Up-Regulates Cyclooxygenase-2 and Prostaglandin E_2 Levels in Human Peritoneal Fibroblasts*. 60th Annual Meeting of the American Society for Reproductive Medicine, Philadelphia, PA, October 2004.

45. *Dichloroacetate Inhibition of Angiogenesis Caused by Hypoxia Treatment of Normal Peritoneal and Adhesion Fibroblasts in Human Umbilical Vein Endothelial Cells*. 60th Annual Meeting of the American Society for Reproductive Medicine, Philadelphia, PA, October 2004.

46. *Dichloroacetate Significantly Increase the Expression of the Transcription Nuclear Factor Kappa-β in Fibroblasts of Human Adhesion Tissues*. 51st Annual Scientific Meeting of the Society for Gynecologic Investigation, Houston, TX, March 2004.

47. *Stimulation of Expression of Vascular Endothelial Growth Factor by Hypoxia from Fibroblasts Isolated from Normal Peritoneum and Adhesion Tissues*. 32nd Annual Meeting of The American Association of Gynecologic Laparoscopists, Las Vegas, NV, November 2003.

48. *Inhibition of Nitric Oxide Production by N-Nitro-L-Arginine Methyl Ester Increased the Expression of Type I Collagen in Human Peritoneal Fibroblasts*. 59th Annual Meeting of American Society for Reproductive Medicine, San Antonio, TX, October 2003.

49. *Apoptosis of Human Peritoneal and Adhesion Fibroblasts After Hypoxia: Role of Inducible Nitric Oxide Synthase*. 59th Annual Meeting of American Society for Reproductive Medicine, San Antonio, TX, October 2003.

50. *Inhibition of Cyclooxygenase-2 in Fibroblasts Isolated from Normal Peritoneum and Adhesion Tissues Decreases the Expression of Hypoxia Inducible Factor-1 Alpha*. 50th Annual Scientific Meeting of the Society for Gynecologic Investigation, Washington, DC, March 2003.

51. *Tissue Plasminogen Activator/Plasminogen Activator Inhibitor-1 (tPA/PAI-1) Modulation by Tisseel*. 50th Annual Scientific Meeting of the Society for Gynecologic Investigation, Washington, DC, March 2003.

52. *Hypoxia Increases the Expression of Vascular Endothelial Growth Factor in Fibroblasts Isolated From Human Normal Peritoneum and Adhesion Tissues*. 50th Annual Scientific Meeting of the Society for Gynecologic Investigation, Washington, DC, March 2003.

53. *Dichloroacetate Significantly Reduces the Expression of Vascular Endothelial Growth Factor in Fibroblasts of Human Adhesion Tissues*. 50th Annual Scientific Meeting of the Society for Gynecologic Investigation, Washington, DC, March 2003.

54. *Transforming Growth Factor-Beta 1 (TGF-β1) and Extracellular Matrix Production by Human Peritoneal Mesothelial Cells: Effect of Tisseel® Fibrin Sealant)*. 50th Annual Scientific Meeting of the Society for Gynecologic Investigation, Washington, DC, March 2003.

55. *Cyclooxygenase-2 Inhibition Decreases the Expression of Vascular Endothelial Growth Factor from Fibroblasts Isolated from Normal Peritoneum and Adhesion Tissues*. 50th Annual Scientific Meeting of the Society for Gynecologic Investigation, Washington, DC, March 2003.

56. *Elevation of Type I Collagen mRNA in Peritoneal Adhesions*. 31st Annual Meeting of the American Association of Gynecologic Laparoscopists, Miami, FL, November 2002.

57. *Cyclooxygenase-2 Expression in Human Fibroblasts Isolated from Adhesions But Not from Normal Peritoneal Tissues*. 31st Annual Meeting of the American Association of Gynecologic Laparoscopists, Miami, FL, November 2002.

58. *Existence of p53 Expression in Human Fibroblasts Isolated from Adhesions, But Not from Normal Peritoneal Tissues*. 31st Annual Meeting of the American Association of Gynecologic Laparoscopists, Miami, FL, November 2002.

59. *Matrix Metalloproteinase (MMP-1, MMP-2), and Tissue Inhibitor for Metalloproteinase (TIMP-1) Expression by Human Peritoneal Mesothelial Cells: Effect of Fibrin Sealant*. 58th Annual Meeting of the American Society for Reproductive Medicine, Seattle, WA, October 2002.

60. *Dichloroacetate (DCA) Significantly Increases the Expression of Inducible Nitric Oxide Synthase (INOS) in Human Fibroblasts of Adhesion Tissues, But Not In Normal Peritoneum*. 58th Annual Meeting of the Society for Reproductive Medicine, Seattle, WA, October 2002.

61. *Seprafilm (Modified Hyaluronic Acid Carboxymethylcellulose) Acts as a Mechanical Barrier*. 49th Scientific Meeting of the Society for Gynecologic Investigation, Los Angeles, CA, March 2002.

62. *Inhibition of Cyclooxygenase-2 in Human Adhesion Fibroblasts Reduces the Expression of MMP-1 and TIMP-1*. 49th Scientific Meeting of the Society for Gynecologic Investigation, Los Angeles, CA, March 2002.

63. *Inhibition of Cyclooxygenase-2 in Human Adhesion Fibroblasts Reduces the Expression of Transforming Growth Factor Beta-1*. 49th Scientific Meeting of the Society for Gynecologic Investigation, Los Angeles, CA, March 2002.

64. *Adhesion Phenotype: Cyclooxygenase-2 is Expressed in Fibroblasts Isolated From Adhesions, But Not From Normal Peritoneal Tissues*. 49th Scientific Meeting of the Society for Gynecologic Investigation, Los Angeles, CA, March 2002.

65. *Reduction of the Expression of Type I and III Collagens in Human Adhesion Fibroblasts, But Not in Normal Peritoneal Fibroblasts by the Inhibition of Cyclooxygenase-2*. 49th Scientific Meeting of the Society for Gynecologic Investigation, Los Angeles, CA, March 2002.

66. *Dichloroacetate Significantly Reduces the Expression of Cyclooxygenase-2 in Human Fibroblasts of Adhesion Tissues*. 49th Scientific Meeting of the Society for Gynecologic Investigation, Los Angeles, CA, March 2002.

67. *Adhesion Phenotype: p53 is Expressed in Fibroblasts Isolated From Adhesions But Not From Normal Peritoneal Tissues*. 49th Scientific Meeting of the Society for Gynecologic Investigation, Los Angeles, CA, March 2002.

68. *Metabolic Regulation of Collagen I in Fibroblasts Isolated from Normal Peritoneum and Adhesions by Dichloroacetic Acid (DCA)*. 28th Scientific Meeting of Gynecologic Surgeons, Dallas, TX, March 2002.

69. *An Adhesion Promoting Phenotype: Implications for Postoperative Adhesion Development*. 30th Annual Meeting American Association of Gynecologic Laparoscopists, Global Congress of Gynecologic Endoscopy, San Francisco, CA, November 2001.

70. *Differences in the Rate of Apoptosis Following Hypoxia in Human Peritoneal and Adhesion Fibroblasts*. 30th Annual Meeting American Association of Gynecologic Laparoscopists, Global Congress of Gynecologic Endoscopy, San Francisco, CA, November 2001.

71. *Modulation of the BCL-2/BAX Ratio by IFN-GAMMA and Hypoxia in Human Peritoneal and Adhesion Fibroblasts*. 57th Annual Meeting of the American Society for Reproductive Medicine, Orlando, FL, October 2001.

72. *Significance of the Effect of Hypoxia on the Rate of Apoptosis of Human Peritoneal and Adhesion Fibroblasts for Postoperative Adhesion Development*. 57th Annual Meeting of the American Society for Reproductive Medicine, Orlando, FL, October 2001.

73. *Prostaglandin E_2 Stimulates Proliferation and Reduces Apoptosis in Epithelial Ovarian Cancer Cell Lines*. 48th Annual Meeting of the Society for Gynecologic Investigation, Toronto, Canada, March 2001.

74. *Differential Modulation of BCL-2/BAX Ratio by Hypoxia in Peritoneal and Adhesion Fibroblasts Cultured from the Same Patient*. 48th Annual Meeting of the Society for Gynecologic Investigation, Toronto, Canada, March 2001.

75. *Interferon Gamma Blocks the Stimulating Effect of Hypoxia on the Expression of Type I Collagen and Fibronectin in Human Peritoneal and Adhesion Fibroblasts*. 48th Annual Meeting of the Society for Gynecologic Investigation, Toronto, Canada, March 2001.

76. *The Effect of Interferon Gamma and Hypoxia on the Expression of TGF-β Isoforms in Human Peritoneal and Adhesion Fibroblasts*. 48th Annual Meeting of the Society for Gynecologic Investigation, Toronto, Canada, March 2001.

77. *The Effect of Normoxia after Hypoxia Treatment of the Expression of Type I Collagen and TGF-β1 in Human Peritoneal Fibroblasts: Implications for Postoperative Adhesion Development*. 48th Annual Meeting of the Society for Gynecologic Investigation, Toronto, Canada, March 2001.

78. *Modulation of the BCL-2/BAX Ratio by IFN-γ and Hypoxia in Human Peritoneal and Adhesion Fibroblasts*. 32nd Annual Meeting of the Society of Gynecologic Oncologists, Nashville, TN, February 2001.

79. *Prostaglandin in Induced COX-2 Expression and Reduced Apopotosis in Epithelial Ovarian Cancer Cells*. 32nd Annual Meeting of the Society of Gynecologic Oncologists, Nashville, TN, February 2001.

80. *The Effect of Hypoxia on the Expression of HIF-1β, BAX, and BCL-2 in the Epithelial Ovarian Cancer Cell Line MADH2774*. 32nd Annual Meeting of the Society of Gynecologic Oncologists, Nashville, TN, February 2001.

81. *Induction of Cyclooxygenase-2 by Prostaglandin E_2 in Human Ovarian Cancer Cell Lines*. 53rd Congress of the DGGG, German Society of Gynecology and Obstetrics eV, Munich, Germany, June 2000.

82. *Type I Collagen Production by Human Peritoneal Fibroblasts in Response to Hypoxia and/or Transforming Growth Factor-Beta 1 (TGF-β1) Treatments*. 47th Annual Meeting of the Society for Gynecologic Investigation, SGI 2000-A Millennial Milestone in Reproductive Sciences: Celebrating the Promise, Chicago, IL, March 2000.

83. *The Effect of Hypoxia on TGF-β1 on the Expression of Cellular Fibronectin in Human Peritoneal Fibroblast Cells in Culture*. 47th Annual Meeting of the Society for Gynecologic Investigation, SGI 2000-A Millennial Milestone in Reproductive Sciences: Celebrating the Promise, Chicago, IL, March 2000.

84. *Type I Collagen Expression in Adhesion and Normal Peritoneal Tissues*. 47th Annual Meeting of the Society for Gynecologic Investigation, SGI 2000-A Millennial Milestone in Reproductive Sciences: Celebrating the Promise, Chicago, IL, March 2000.

85. *Vascular Endothelial Growth Factor (VEGF) Levels Are Elevated in Adhesion Tissue in Humans*. Annual Meeting of the American Association of Gynecologic Laparoscopists, Las Vegas, NV, November 1999.

86. *Basics of Cutaneous Wound Repair*. 4th International Conference on Postoperative Healing and Adhesions, Fort Lauderdale, FL, October 1999.

87. *The Role of Extracellular Matrix in the Formation of Postoperative Adhesion*. 4th International Conference on Postoperative Healing and Adhesions, Fort Lauderdale, FL, October 1999.

88. *The Effect of Hypoxia and TGF-β1 on the Expression of Tissue Inhibitors of Metalloproteinases (TIMP-1) in Human Peritoneal Mesothelial Cells*. Joint meeting of the Canadian Fertility Society and the American Society for Reproductive Medicine, Toronto, Ontario, Canada, September 1999.

89. *Collagen Type I and Type III Production by Human Mesothelial Cells in Response to Hypoxia and/or TGF-β1 Treatments*. Annual Meeting of the Society for Gynecologic Investigation, Atlanta, GA, March 1999.

90. *The Role of Apoptosis and p53 in the Pathogenesis of Keloids*. Journal of Investigative Dermatology 110: 597, 1998.

91. *Apoptosis Modulation in the Response of CTCL to PUVA*. Journal of Investigative Dermatology 110: 698, 1998.

92. *Apoptosis Dysregulation in Keloid Fibroblasts*. Journal of Investigative Dermatology 110:653, 1998.

93. *Apoptosis Regulation in the Pathogenesis of Cutaneous T-Cell Lymphoma (CTCL)*. Journal of Investigative Dermatology 108:610, 1997.

94. *The Effect of PUVA Treatment on HUT78 Cell Differential Gene Expression*. Journal of Investigative Dermatology 106:906, 1996.

95. *Detection of Differentially Displayed cDNA Fragments in Normal vs Sezary Syndrome Leukocytes*. Journal of Investigative Dermatology 104: 673, 1995.

96. *Quantitative PCR Analysis of Th-1 Cytokines in HUT78 Cells after Exposure to PUVA In Vitro*. Journal of Investigative Dermatology 102: 585, 1994.

97. *Augmentation of Th-1 Cytokines in the Peripheral Blood of Sezary Syndrome Patients after Treatment with ECCP*. Journal of Investigative Dermatology 102:586, 1994.

98. *Augmentation of Th-1 Cytokines in the Peripheral Blood of SZ Patients Upon Treatment with Extracorporeal Photopheresis*. Clinical Research 41:664, 1993.

99. *Detection of T-Cell Clonality in Mycosis Fungoides by PCR-Metaphore Agarose Analysis of T-Cell Receptor-γ*. Clinical Research 41:459, 1993.

100. *Mycosis Fungoides and Psoriasis Exhibit a Th1 Type Cell Mediated Response While Sezary Syndrome Expresses A Th2 Type Response*. Clinical Research 40:730, 1992.

101. *T-Cell Receptor Gene Conservation and Rearranged Clones in Canine Mycosis Fungoides*. Clinical Research 40:505, 1992.

Poster Presentations (Referred)

1. Fletcher NM, Awonuga AO, Memaj I, Diamond MP, **Saed GM**. Interruption of MPO Binding to CD11B Selectively Kills Fibroblasts from Adhesion Tissues but not Normal Peritoneum. 73rd American Society for Reproductive Medicine Scientific Congress & Expo, San Antonio, TX, October-November 2017. Proceedings: P-264, 216, 2017. **SRS In-Training Award for Research to NM Fletcher, PhD**

2. Fletcher NM, Memaj I, Abusamaan MS, Juhani A, Al-Hendy A, Diamond MP, **Saed GM**. Oxidative Stress: A Key Regulator of Leiomyoma Cell Survival. 64th Annual Scientific Meeting for the Society for Reproductive Investigation, Orlando, FL, March 2017. Fertility and Sterility 24(1) Supplement: F-124, 208A, 2017.

3. Detti L, Fletcher NM, **Saed GM**, Uhlmann RA, Christiansen ME, Williams LJ. Anti-Mullerian Hormone (AMH) Regulates BRCA1 and BRCA2 Gene Expression in an Ovarian Cortex Transplantation Model. 72nd Annual Meeting of the American Society for Reproductive Medicine, Salt Lake City, UT, October 2016. Fertility and Sterility 106(3) Supplement: P-037, e120, 2016.

4. Fletcher NM, Belotte J, Saed MG, Abusamaan MS, Diamond MP, **Saed GM**. Chemotherapy Induces a Genotype Switch in Key Antioxidant Enzymes: A Potential Mechanism of Chemoresistance in Epithelial Ovarian Cancer Cells. 63rd Annual Meeting of the Society for Reproductive Investigation, Montreal, Quebec, Canada, March 2016. Reproductive Sciences 23(1) Supplement: F-248, 262-263A, 2016.

5. Detti L, Fletcher NM, Uhlmann RA, Belotte J, Williams LJ, **Saed GM**. Exposure to Recombinant Anti-Mullerian Hormone (AMH) Downregulates Ovarian Follicle Cells' Stemness Potential in Fresh and Vitrified/Thaw Ovarian Cortex. 63rd Annual Meeting of the Society for Reproductive Investigation, Montreal, Quebec, Canada, March 16-19, 2016. Reproductive Sciences 23(1) Supplement: T-257, 180A, 2016.

6. Nusrat O, Belotte J, Fletcher NM, Saed MG, Diamond MP, **Saed GM**. Chemoresistant Ovarian Cancer Cells Manifest Lower Vascular Endothelial Growth Factor and Hypoxia Inducible Factor-1α: A Potential Survival Mechanism. 63rd Annual Meeting of the Society for Reproductive Investigation, Montreal, Quebec, Canada, March 16-19, 2016. Reproductive Sciences 23(1) Supplement: T-250, 178A, 2016.

7. Fletcher NM, Neubauer BR, Saed MG, Abu-Soud HM, **Saed GM**. 2,4-Dinitrophenol Induced Cell Death of Ovarian Cancer Stem Cells. 62nd Annual Meeting of the Society for Reproductive Investigation, San Francisco, CA, March 2015. Reproductive Sciences 22(1) Supplement: S-003, 299A, 2015.

8. Fletcher NM, Neubauer BR, Saed MG, Diamond MP, Abu-Soud HM, **Saed GM**. Postoperative Adhesion Development is Controlled by Mechanisms Emanating from a Hypoxia-Induced Genotype Switch in Nicotinamide Adenine Dinucleotide Phosphate Oxidase Through the Up-Regulation of Cytidine Deaminase. 62nd Annual Meeting of the Society for Reproductive Investigation, San Francisco, CA, March 2015. Reproductive Sciences 22(1) Supplement: F-042, 218A, 2015.

9. Detti L, Williams LJ, Fletcher NM, **Saed GM**. Anti-Müllerian Hormone (AMH) May Inhibit Oocyte Maturation and Follicular Vascularization in Human Ovarian Cortex. Proceedings of the 71st Annual Meeting of the American Society for Reproductive Medicine, Baltimore, MD, October 2015. Fertility and Sterility 104(3) Supplement: P91, e136, 2015.

10. Fletcher NM, Saed MG, Neubauer BR, Abusamaan MS, Al-Hendy A, Diamond MP, Berman JM, **Saed GM**. Uterine Fibroids Are Characterized by An Altered Redox Balance, Favoring A Pro-Oxidant State. 71st Annual Meeting of the American Society for Reproductive Medicine, Baltimore, MD, October 2015. Fertility and Sterility 104(3) Supplement: P-115, e145, 2015.

11. Fletcher NM, Saed MG, Neubauer BR, Abu-Soud HM, Awonuga A, Diamond MP, **Saed GM**. Shifting Anaerobic to Aerobic Metabolism Stimulates Apoptosis in Adhesion Fibroblasts Through the Modulation of the Cellular Redox Homeostasis. 71st Annual Meeting of the American Society for Reproductive Medicine, Baltimore, MD, October 2015. Fertility and Sterility 104(3) Supplement: P-215, e179, 2015.

12. Abusamaan MS, Fletcher NM, Saed MG, Al-Hendy A, Diamond MP, Berman JM, **Saed GM**. Myeloperoxidase Serves As A Redox Switch That Regulates Apoptosis In Human Leiomyomas. 71st Annual Meeting of the American Society for Reproductive Medicine, Baltimore, MD, October 2015. Fertility and Sterility 104(3) Supplement: P-113, e145, 2015.

13. Fletcher NM, Detti L, Neubauer BR, Saed MG, Diamond MP, Abuzeid MI, **Saed GM**. Altered Redox State in the Endometrium of Patients Undergoing Ovarian Stimulation for Assisted Reproduction Technology. Proceedings of the 70th Annual Meeting of the American Society for Reproductive Medicine, Honolulu, HI, October 2014. Fertility and Sterility 102(35) Supplement: P-426, e279, 2014.

14. Belotte J, Fletcher NM, Diamond MP, **Saed GM**. Sox2 Gene Amplification Impacts Survival in Serous Epithelial Ovarian Cancer. 61st Annual Meeting of the Society for Investigation, Florence, Italy, March 2014. Reproductive Sciences 21(3) Supplement: T-219, 204A, 2014.

15. **Saed GM**, Fletcher NM, Belotte J, Levin NK, Simon MS, Abu-Soud HM, Tainsky MA, Diamond. SNPs in Key Oxidants and Antioxidants Are Associated with Increased Risk and Serve as Potential Targets for Ovarian Cancer. 61st Annual Meeting of the Society for Gynecologic Investigation, Florence, Italy, March 2014. Reproductive Sciences 21(3) Supplement: T-249, 213A, 2014.

16. Diamond MP, Fletcher NM, Saed MG, Abu-Soud HM, Al-Hendy A, **Saed GM**. Fibroids Manifest Oxidative Stress As Compared to Normal Myometrium. 42nd Annual AAGL Global Congress of Minimally Invasive Gynecology, Washington, DC, November 2013. The Journal of Minimally Invasive Gynecology 20(6) Suppl: S19, 2013.

17. Diamond MP, Fletcher NM, Abuanzeh S, Saed MG, **Saed GM**. Creation and Persistence of the Adhesion Phenotype: The Role of NOXs in Creating Oxidative Stress. 42nd Annual AAGL Global Congress of Minimally Invasive Gynecology, Washington, DC, November 2013.

18. Fletcher NM, Saed MG, Abu-Soud HM, Al-Hendy A, Diamond MP, **Saed GM**. Distinct Oxidative Stress Profile in Uterine Fibroids Versus Adjacent Myometrium. Conjoint Meeting of the International Federation of Fertility Societies and the 69th American Society for Reproductive Medicine, Boston, MA, October 2013. Fertility and Sterility 100(3) Suppl: S34, 2013.

19. Fletcher NM, Abuanzeh S, Saed MG, Abu-Soud HM, Diamond MP, **Saed GM**. Postoperative Adhesion is Characterized by a Unique Oxidative Stress Profile Which is Responsible for Creation and Persistence of the Adhesion Phenotype. Conjoint Meeting of the International Federation of Fertility Societies and the 69th American Society for Reproductive Medicine, Boston, MA, October 2013. Fertility and Sterility 100(3) Suppl: S31, 2013.

20. Thakur M, Imudia AN, Shavell VI, Singh M, Diamond MP, Awonuga AO, **Saed GM**. Should Body Mass Index Influence the Dose of hCG for Ovulation Induction After Superovulation in IVF/ICSI cycles? 68th Annual Meeting of the American Society for Reproductive Medicine, San Diego, CA, October 2012. Fertility and Sterility 98(3) Suppl: P-542, S271, 2012.

21. Fletcher NM, Al-Hendy A, Diamond M, **Saed GM**. Uterine Fibroids Are Characterized by an Impaired Antioxidant Cellular System: Potential Role of Hypoxia in the Pathophysiology of Fibroids. 68th Annual Meeting of the American Society for Reproductive Medicine, San Diego, CA, October 2012. Fertility and Sterility 98(3) Suppl: P-403, S231, 2012.

22. Detti L, Uhlmann RA, Fletcher NM, Diamond MP, **Saed GM**. Endometrial Thyroid and Vitamin D Signaling Pathways during Ovarian Stimulation for Assisted Reproductive Technology (ART). 68th Annual Meeting of the American Society for Reproductive Medicine, San Diego, CA, October 2012. Fertility and Sterility 98(3) Suppl: P-384, S225, 2012.

23. Fletcher NM, Belotte J, Diamond MP, **Saed GM**. Dicholoroacetate Increases Sensitivity to Chemotherapy Treatment of Epithelial Ovarian Cancer Cells. 59th Annual Meeting of the Society for Gynecologic Investigation, San Diego, CA, March 2012. Reproductive Sciences 19(3) Suppl: S-065, 354A, 2012.

24. Banerjee J, Maitra D, Shaeib F, **Saed GM**, Diamond MP, Abu-Soud HM. Melatonin Prevents Hypochlorous Acid Induced Alteration of the Metaphase-II Mouse Oocyte Microtubule and Chromosomal Structure. 59th Annual Meeting of the Society for Gynecologic Investigation, San Diego, CA, March 2012. Reproductive Sciences 19(3) Suppl: E-212, 289A, 2012.

25. **Saed GM**, Fletcher NM, Ruden DM, Abu-Soud HM, Diamond MP. Epigenetics: New Insights into Postoperative Adhesion Development. 59th Annual Meeting of the Society for Gynecologic Investigation, San Diego, CA, March 2012.

26. Nair S, **Saed GM**, Atta HM, Diamond M, Al-Hendy A. Gene Therapy of Abdominal/Pelvic Post-Operative Adhesions: Targeting Adenovirus towards Human Peritoneal Adhesion Cells. 59th Annual Meeting of the Society for Gynecologic Investigation, San Diego, CA, March 2012. Reproductive Sciences 19(3) Suppl: T-066, 141A, 2012

27. Banerjee J, Maitra D, Shaeib F, **Saed GM**, Diamond MP, Abu-Soud H. Role of Melatonin in Preventing Hypochlorous Acid Induced Alterations in Microtubule and Chromosomal Structure in Metaphase-II Mouse Oocytes *In Vitro*. 67th Annual Meeting of the American Society for Reproductive Medicine, Orlando, FL, October 2011. Fertility and Sterility (Suppl 1): P-450, 2011.

28. Abu-Farsakh SM, Abu-Farsakh HM, Fletcher NM, **Saed GM**, Diamond MP. Histopathologic Analysis in Testicular Azoospermia. 67th Annual Meeting of the American Society for Reproductive Medicine, Orlando, FL, October 2011. Fertility and Sterility (Suppl 1): P-183, 2011.

29. Shavell VI, Fletcher NM, Jiang ZL, **Saed GM**, Diamond MP. Uncoupling Oxidative Phosphorylation with 2,4-Dinitrophenol Promotes Development of the Adhesion Phenotype. 67th Annual Meeting of the American Society for Reproductive Medicine, Orlando, FL, October 2011. Fertility and Sterility (Suppl 1): P-131, 2011.

30. Nair S, **Saed G**, Nwaobasi N, Atta H, Al-Hendy A. Towards Gene Therapy of Pelvic Post-Operative Adhesions: Targeting Adenovirus Towards Human Adhesion Cells. 67th Annual Meeting of the American Society for Reproductive Medicine, Orlando, FL, October 2011. Fertility and Sterlity (Suppl 1): P-111, 2011.

31. Detti L, **Saed GM**, Fletcher NM, Kruger ML, Brossoit B, Diamond MP. Endometrial Morphology and Modulation of Hormone Receptors during Ovarian Stimulation for Assisted Reproductive Technology Cycles. 66th Annual Meeting of the American Society for Reproductive Medicine, Denver, CO, October 2010. Fertility and Sterility 94(4) Suppl 1: S213-S214, 2010.

32. Fletcher NM, Jiang Zl, Almahmoud H, Diamond MP, **Saed GM**. Human Adhesion Fibroblasts Are Under Constant intrinsic oxidative stress as characterized by higher baseline NADPH oxidase and hypoxia inducible factor- 1α and lower baseline superoxide dismutase. 66th Annual Meeting of the American Society for Reproductive Medicine, Denver, CO, October 2010. Fertility and Sterility 94(4) Suppl 1: S208, 2010.

33. White J, Jiang Z, Diamond M, **Saed G**. The role of macrophages in the development of the adhesion phenotype. Proceedings of the 66th Annual Meeting of the American Society for Reproductive Medicine, Denver, CO, October 2010. Fertility and Sterility 94(4) Suppl 1: S202, 2010.

34. Huang K, **Saed GM**, Crispino J, Song J, Choi SD, Diamond M, Naftolin F. Membrane-actin cytoskeleton linking protein expression by human post-operative adhesions and fibroblasts. 57th Annual Meeting of the Society for Gynecologic Investigation, Orlando, FL, March 2010. Reproductive Sciences 17(3) Suppl: P655, 253A, 2010.

35. **Saed GM**, Jiang ZL, Fletcher NM, Al Arab A, Abu-Soud HM, Munkarah AM, Diamond MP. Dichloroacetate induces apoptosis of epithelial ovarian cancer cells through the inhibition of oxidative stress enzymes. Proceedings of the 57th Annual Meeting of the Society for Gynecologic Investigation, Orlando, FL, March 2010. Reproductive Sciences 17(3) Suppl: P171, 113A, 2010.

36. **Saed GM**, Jiang ZL, Fletcher NM, Ali-Fehmi R, Diamond MP, Abu-Soud HM, Munkarah AR. Inhibition of NADPH oxidative reductase promotes apoptosis in epithelial ovarian cancer cells. Proceedings of the 57th Annual Meeting of the Society for Gynecologic Investigation, Orlando, FL, March 2010. Reproductive Sciences 17(3) Suppl: P170, 113A, March 2010.

37. Meng Q, Sun W, Jiang ZL, Fletcher NM, **Saed GM**, Diamond MP. Endometriotic implants resemble ovarian cancer in their inflammatory cytokines and hormone receptors expression: potential transformation into ovarian cancer. Proceedings of the 57th Annual Meeting of the Society for Gynecologic Investigation, Orlando, FL, March 2010. Reproductive Sciences 17(3) Suppl: P97, 93A, 2010.

38. **Saed GM**, Jiang ZL, Fletcher NM, Abu-Soud HM, Diamond MP. Polychlorinated biphenyl congeners induce the adhesion phenotype by reducing superoxide dismutase levels. 65th Annual Meeting of the American Society for Reproductive Medicine, Atlanta, GA, October 2009. Fertility and Sterility 90(Suppl.1): P179, October 2009.

39. **Saed GM**, Hall DT, Omar MW, Shavell VI, Fletcher NM, Diamond MP. Regulation of metabolic activity of peritoneal fibroblasts by dichloroacetate provides a potential target for interventions to reduce postoperative adhesions. 65th Annual Meeting of the American Society for Reproductive Medicine, Atlanta, GA, October 2009. Fertility and Sterility 90(Suppl. 1): P127, October 2009.

40. White J, Jiang Z, Diamond M, **Saed G**. Hypoxia induces transforming growth factor beta 1 (TGFβ1) in human macrophages through a hypoxia inducible factor 1α (HIF-1α) – dependent mechanism. 65th Annual Meeting of the American Society for Reproductive Medicine, Atlanta, GA, October 2009. Fertility and Sterility 90(Suppl. 1): P126, October 2009.

41. **Saed GM**, Fletcher NM, Jiang ZL, Abu-Soud HM, Diamond MP. Sensitivity of human normal peritoneal and adhesion fibroblasts to hypoxia-induced lipid peroxidation depends on the bioavailability of hydrogen peroxide. 56th Annual Scientific Meeting of the Society for Gynecologic Investigation, Glasgow, Scotland, United Kingdom, March 2009. Reproductive Sciences 16(3) Suppl: P1004, 359A, 2009.

42. Abu-Soud HM, Jiang ZL, Fletcher NM, Diamond MP, **Saed GM**. Exposure to polychlorinated biphenyls enhances lipid peroxidation in human normal peritoneal and adhesion fibroblasts: a potential role for MPO. 56th Annual Scientific Meeting of the Society for Gynecologic Investigation, Glasgow, Scotland, United Kingdom, March 2009. Reproductive Sciences 16(3) Suppl: P985, 354A, 2009.

43. Jiang ZL, Fletcher NM, Malone JM Jr, Ali R, Munkarah AR, Diamond MP, Abu-Soud HM, **Saed GM**. NADPH oxidase inhibition attenuates oxidative stress in epithelial ovarian cancer. 56th Annual Scientific Meeting of the Society for Gynecologic Investigation, Glasgow, Scotland, United Kingdom, March 2009. Reproductive Sciences 16(3) Suppl: P291, 153A, 2009.

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8. Fletcher NM, Belotte J, Saed MG, Abu-Soud HM, Diamond MP, **Saed GM**. Dicholoroacetate increases sensitivity to chemotherapy by modulation of antioxidants in epithelial ovarian cancer. Proceedings of the Graduate Research Day, Wayne Day 2014 Program, Current Concepts in Gynecologic Oncology for the Obstetrician and Gynecologist, Kamran S. Moghissi, M.D. Lecture, Department of Obstetrics and Gynecology, Wayne State University School of Medicine, Detroit, MI, December 2014.

9. Shaeib F, Khan SN, Banerjee J, Thakur M, Dai J, Awonuga AO, **Saed GM**, Abu-Soud HM. Role of cumulus cells in defense against reactive oxygen species insult in metaphase II mouse oocytes. 4th Annual Scientific Retreat of The C.S. Mott Center for Human Growth and Development, Department of Obstetrics and Gynecology, Wayne State University School of Medicine, Detroit, MI, May 2014. Program and Abstracts: #P-26, pg. 39, 2014.

10. Nusrat O, Fletcher NM, Belotte J, Saed MG, Neubauer BR, **Saed GM**. Chemoresistant ovarian cancer cells manifest lower vascular endothelial growth factor and hypoxia induced factor-1α: a potential survival mechanism. 4th Annual Scientific Retreat of The C.S. Mott Center for Human Growth and Development, Department of Obstetrics and Gynecology, Wayne State University School of Medicine, Detroit, MI, May 2014. Program and Abstracts: #P-23, pg. 36, 2014.

11. Najafi T, Goud PT, Goud AP, Gonik B, **Saed GM**, Zhang X, Abu-Soud HM. Direct real-time measurement of intra-oocyte nitric oxide concentration *in vivo*. 4th Annual Scientific Retreat of The C.S. Mott Center for Human Growth and Development, Department of Obstetrics and Gynecology, Wayne State University School of Medicine, Detroit, MI, May 2014; Program and Abstracts: #P-22, pg. 35, 2014.

12. Fletcher NM, Belotte J, Saed MG, Abu-Soud HM, Diamond MP, **Saed GM**. Dicholoroacetate increases sensitivity to chemotherapy by modulation of antioxidants in epithelial ovarian cancer. 4th Annual Scientific Retreat of The C.S. Mott Center for Human Growth and Development, Department of Obstetrics and Gynecology, Wayne State University School of Medicine, Detroit, MI, May 2014. Program and Abstracts: #P-07, pg. 20, 2014.

13. Belotte J, Fletcher NM, Saed MG, Neubauer BR, **Saed GM**. RAD21 gene amplification impacts survival in serous epithelial ovarian cancer. 4th Annual Scientific Retreat of The C.S. Mott Center for Human Growth and Development, Department of Obstetrics and Gynecology, Wayne State University School of Medicine, Detroit, MI, May 2014. Program and Abstracts: #P-04, pg. 17, 2014.

14. Belotte J, Fletcher NM, Nusrat O, Saed MG, Neubauer BR, Abu-Soud HM, **Saed GM**. Superoxide dismutase significantly delayed the development of cisplatin resistance in epithelial ovarian cancer cells. 4th Annual Scientific Retreat of The C.S. Mott Center for Human Growth and Development, Department of Obstetrics and Gynecology, Wayne State University School of Medicine, Detroit, MI, May 2014. Program and Abstracts: #P-03, pg. 16, 2014.

15. Awonuga AO, Fletcher NM, Belotte J, Diamond MP, **Saed GM**. The in-vivo effects of superoxide dismutase on the incidence and severity of postoperative adhesion development. 4th Annual Scientific Retreat of The C.S. Mott Center for Human Growth and Development, Department of Obstetrics and Gynecology, Wayne State University School of Medicine, Detroit, MI, May 2014. Program and Abstracts: #P-02, pg. 15, 2014.

16. **Saed GM**. The role of oxidative stress in the pathogenesis of pro-fibrotic gynecologic disorders. 4th Annual Scientific Retreat of The C.S. Mott Center for Human Growth and Development, Department of Obstetrics and Gynecology, Wayne State University School of Medicine, Detroit, MI, May 2014. Program and Abstracts: #O-13, pg. 13, 2014.

17. Najafi T, Goud AP, Goud PT, **Saed GM**, Gonik B, Abu-Soud HM. Release of substrates, cofactors, and products of nitric oxide synthase are altered during oocyte aging. 4th Annual Scientific Retreat of The C.S. Mott Center for Human Growth and Development, Department of Obstetrics and Gynecology, Wayne State University School of Medicine, Detroit, MI, May 2014. Program and Abstracts: #O-12, pg. 12, 2014.

18. Shaeib F, Khan SN, Ali I, Dai J, Drewlo S, **Saed GM**, Abu-Soud HM. The impact of myeloperoxidase on metaphase II mouse oocyte quality. 4th Annual Scientific Retreat of The C.S. Mott Center for Human Growth and Development, Department of Obstetrics and Gynecology, Wayne State University School of Medicine, Detroit, MI, May 2014. Program and Abstracts: #O-11, pg. 11, 2014.

19. Fletcher NM, **Saed GM**. Differential expression of glutathione peroxidase and glutathione reductase in chemoresistant epithelial ovarian cancer cells. 4th Annual Research Symposium of the Michigan Alliance for Reproductive Technologies and Science (MARTS), University of Michigan, Ann Arbor, MI, May 2013.

20. Shaeib F, Banerjee J, Thakur M, Saed MG, Diamond MP, **Saed GM**, Abu-Soud HM. Confocal 3-dimensional reconstruction can serve as a useful tool to quantify oxidative stress induced oocyte spindle damage. 3rd Annual Scientific Retreat of The C.S. Mott Center for Human Growth and Development, Department of Obstetrics and Gynecology, Wayne State University School of Medicine, Detroit, MI, May 15, 2013. Program and Abstracts: #35, 2013.

21. Fletcher NM, **Saed GM**. Differential expression of glutathione peroxidase and glutathione reductase in chemoresistant epithelial ovarian cancer cells. 3rd Annual Scientific Retreat of The C.S. Mott Center for Human Growth and Development, Department of Obstetrics and Gynecology, Wayne State University School of Medicine, Detroit, MI, May 2013: Program and Abstracts: #26, 2013.

22. **Saed GM**. Investigation of the role of oxidative stress in the pathophysiology of gynecologic fibrotic disorders including postoperative adhesions, fibroids, and endometriosis as well as ovarian cancer. 3rd Annual Scientific Retreat of The C.S. Mott Center for Human Growth and Development, Department of Obstetrics and Gynecology, Wayne State University School of Medicine, Detroit, MI, May 2013. Program and Abstracts: #20, 2013.

23. Belotte J, Mitchell A, Belotte J, **Saed GM**. Sox2 gene copy number alteration (CAN) significantly impact overall survival (OS) in serous epithelial ovarian cancer. 3rd Annual Scientific Retreat of The C.S. Mott Center for Human Growth and Development, Department of Obstetrics and Gynecology, Wayne State University School of Medicine, Detroit, MI, May 2013. Program and Abstracts: #6, 2013.

24. Belotte J, Fletcher NM, Abuanzeh S, Levin NK, Simon NS, Diamond MP, Abu-Soud HM, Tainsky MA, **Saed GM**. A novel association between a catalase single nucleotide polymorphism and increased risk of ovarian cancer. 3rd Annual Scientific Retreat of The C.S. Mott Center for Human Growth and Development, Department of Obstetrics and Gynecology, Wayne State University School of Medicine, Detroit, MI, May 2013. Program and Abstracts: #4, 2013.

25. Awonuga AO, King NM, Belotte J, Abuanzeh S, Diamond MP, **Saed GM**. The in vitro effects of superoxide dismutase on the incidence and severity of post-operative adhesion development after cecal abrasion. 3rd Annual Scientific Retreat of The C.S. Mott Center for Human Growth and Development, Department of Obstetrics and Gynecology, Wayne State University School of Medicine, Detroit, MI, May 2013. Program and Abstracts: #3, 2013.

26. Shavell VI, Fletcher NM, Abu-Soud HM, Diamond MP, **Saed GM**, Detti L. Superoxide dismutase levels are elevated in the peri-implantation endometrium in women undergoing ovarian stimulation. 3rd Annual Michigan Alliance for Reproductive Technologies and Science Research Symposium, Wayne State University School of Medicine, Detroit, MI, May 2012. Proceedings and Abstracts: #15, 2012.

27. Maitra D, Abdulhamid I, **Saed GM**, Diamond MP, Pennathur S, Abu-Soud HM. Fluorescent heme degradation products in Sickle cell disease: role of hypochlorous acid in hemoglobin destruction. 3rd Annual Michigan Alliance for Reproductive Technologies and Science Research Symposium, Wayne State University School of Medicine, Detroit, MI, May 2012. Proceedings and Abstracts: #12, 2012.

28. Maitra D, Abdulridha RM, Byun J, Souza CEA, Banerjee J, Andreana PR, Diamond MP, **Saed GM**, Pennathur S, Abu-Soud. The reaction of HOCl and cyanocobalamin: corrin destruction and the liberation of cyanogens chloride. 3rd Annual Michigan Alliance for Reproductive Technologies and Science Research Symposium, Wayne State University School of Medicine, Detroit, MI, May 2012. Program and Abstracts: #11, 2012.

29. Fletcher NM, Belotte J, Diamond MP, **Saed GM**. Dicholoroacetate increases sensitivity to chemotherapy treatment of epithelial ovarian cancer cells. 3rd Annual Michigan Alliance for Reproductive Technologies and Science Research Symposium, Wayne State University School of Medicine, Detroit, MI, May 2012. Program and Abstracts: #8, 2012.

30. Belotte J, Fletcher NM, Diamond MP, **Saed GM**. The role of oxidative stress in the development of cisplatin resistance in epithelial ovarian cancer. 3rd Annual Michigan Alliance for Reproductive Technologies and Science Research Symposium, Wayne State University School of Medicine, Detroit, MI, May 2012. Program and Abstracts: #6, 2012.

31. Banerjee J, Maitra D, Shaeib F, **Saed GM**, Diamond MP, Abu-Soud HM. Melatonin prevents hypochlorous acid induced alteration of the metaphase-II mouse oocyte microtubule and chromosomal structure. 3rd Annual Michigan Alliance for Reproductive Technologies and Science Research Symposium, Wayne State University School of Medicine, Detroit, MI, May 2012. Program and Abstracts: #5, 2012.

32. Maitra D, Shaeib F, Abdulridha RM, Souza CEA, **Saed GM**, Abu-Soud HM. Modulation of myeloperoxidase activity by self-generated hypochlorous acid. 3rd Annual Michigan Alliance for Reproductive Technologies and Science Research Symposium, Wayne State University School of Medicine, Detroit, MI, May 2012. Program and Abstracts: #1, 2012.

33. Maitra D, Shaeib F, Abdulridha RM, Souza CEA, **Saed GM**, Abu-Soud HM. Modulation of myeloperoxidase activity by self-generated hypochlorous acid. 2nd Annual Scientific Retreat of The C.S. Mott Center for Human Growth and Development, Department of Obstetrics and Gynecology, Wayne State University School of Medicine, Detroit, MI, May 2012. Program and Abstracts: #32, 2012.

34. Fletcher NM, Belotte J, Diamond MP, **Saed GM**. Dicholoroacetate increases sensitivity to chemotherapy treatment of epithelial ovarian cancer cells. 2nd Annual Scientific Retreat of The C.S. Mott Center for Human Growth and Development, Department of Obstetrics and Gynecology, Wayne State University School of Medicine, Detroit, MI, May 2012. Program and Abstracts: #31, 2012.

35. Belotte J, Fletcher NM, Diamond MP, **Saed GM**. The role of oxidative stress in the development of cisplatin resistance in epithelial ovarian cancer. 2nd Annual Scientific Retreat of The C.S. Mott Center for Human Growth and Development, Department of Obstetrics and Gynecology, Wayne State University School of Medicine, Detroit, MI, May 2012. Program and Abstracts: #29, 2012.

36. Banerjee J, Maitra D, Shaeib F, **Saed GM**, Diamond MP, Abu-Soud HM. Melatonin prevents hypochlorous acid induced alteration of the metaphase-II mouse oocyte microtubule and chromosomal structure. 2nd Annual Scientific Retreat of The C.S. Mott Center for Human Growth and Development, Department of Obstetrics and Gynecology, Wayne State University School of Medicine, Detroit, MI, May 2012. Program and Abstracts: #25, 2012.

37. Maitra D, Abdulridha RM, Byun J, Souza CEA, Banerjee J, Andreana PR, Diamond MP, **Saed GM**, Pennathur S, Abu-Soud HM. The reaction of HoCl and cyanocobalamin: corrin destruction and the liberation of cyanogens chloride. 2nd Annual Scientific Retreat of The C.S. Mott Center for Human Growth and Development, Department of Obstetrics and Gynecology, Wayne State University School of Medicine, Detroit, MI, May 2012. Program and Abstracts: #23, 2012.

38. Shavell VI, Fletcher NM, Abu-Soud HM, Diamond MP, **Saed GM**, Detti LL. Superoxide dismutase levels are elevated in the peri-implantation endometrium in women undergoing ovarian stimulation. 2nd Annual Scientific Retreat of The C.S. Mott Center for Human Growth and Development, Department of Obstetrics and Gynecology, Wayne State University School of Medicine, Detroit, MI, May 2012. Programs and Abstracts: #17, 2012.

39. Maitra D, Abdulhamid I, **Saed GM**, Diamond MP, Pennathur S, Abu-Soud HM. Fluorescent heme degradation products in sickle cell disease: role of hypochlorous acid in hemoglobin destruction. 2nd Annual Scientific Retreat of The C.S. Mott Center for Human Growth and Development, Department of Obstetrics and Gynecology, Wayne State University School of Medicine, Detroit, MI, May 2012. Programs and Abstracts: #16, 2012.

40. **Saed GM**. Investigation of the role of oxidative stress in the pathophysiology of gynecologic fibrotic disorders including postoperative adhesions, fibroids, and endometriosis as well as ovarian cancer. 2nd Annual Scientific Retreat of The C.S. Mott Center for Human Growth and Development, Department of Obstetrics and Gynecology, Wayne State University School of Medicine, Detroit, MI, May 2012. Program and Abstracts: #9, 2012.

41. Shavell VI, Fletcher NM, Jiang ZL, **Saed GM**, Diamond MP. Coupling oxidative phosphorylation with 2,4-dinitrophenol promotes development of the adhesion phenotype. 1st Annual Scientific Retreat of The C.S. Mott Center for Human Growth and Development, Department of Obstetrics and Gynecology, Wayne State University School of Medicine, Southfield, MI, May 2011. Program and Abstracts: #26, 2011.

42. **Saed GM**. The role of oxidative stress in the pathophysiology of gynecologic fibrotic disorders including postoperative adhesions, fibroids, and endometriosis, as well as ovarian cancer. 1st Annual Scientific Retreat of The C.S. Mott Center for Human Growth and Development, Department of Obstetrics and Gynecology, Wayne State University School of Medicine, Southfield, MI, May 2011. Program and Abstracts: #24, 2011.

43. Maitra D, Shaeib F, Diamond MP, **Saed GM**, Abu-Soud HM. Melatonin can attenuate HOCl mediated hemolysis, free iron release and heme degradation from hemoglobin. 1st Annual Scientific Retreat of The C.S. Mott Center for Human Growth and Development, Department of Obstetrics and Gynecology, Wayne State University School of Medicine, Southfield, MI, May 2011. Program and Abstracts: #19, 2011.

44. Maitra D, Byun J, Andreana PR, Abdulhamid I, Diamond MP, **Saed GM**, Pennathur S, Abu-Soud HM. Reaction of hemoglobin with HOCl: possible link between free iron accumulation and oxidative stress. 1st Annual Scientific Retreat of The C.S. Mott Center for Human Growth and Development, Department of Obstetrics and Gynecology, Wayne State University School of Medicine, Southfield, MI, May 2011. Program and Abstracts: #18, 2011.

45. Fletcher NM, Jiang ZL, Levin NK, Abu-Soud HM, Munkarah AR, Tainsky MA, Diamond MP, **Saed GM**. Positive correlation between serum myeloperoxidase and free iron levels with stage of ovarian cancer: potential biomarkers for early detection and prognosis of ovarian cancer. 1st Annual Scientific Retreat of The C.S. Mott Center for Human Growth and Development, Department of Obstetrics and Gynecology, Wayne State University School of Medicine, Southfield, MI, May 2011. Program and Abstracts: #13, 2011.

46. Banerjee J, Maitra D, Shaeib F, **Saed GM**, Diamond MP, Abu-Soud HM. Role of melatonin in preventing hypochlorous acid induced alterations in microtubule and chromosomal structure in metaphase-II mouse oocytes *in vitro*. 1st Annual Scientific Retreat of The C.S. Mott Center for Human Growth and Development, Department of Obstetrics and Gynecology, Wayne State University School of Medicine, Southfield, MI, May 2011. Program and Abstracts: #7, 2011.

47. Diamond MP, **Saed GM**. Reduction of postoperative adhesions. Catalyzing Collaboration between Industry and Academic in the Life Sciences – Women's Health Medicine: Part I, Therapeutic Strategies Meeting, Illinois Science and Technology Park, Skokie, IL, June 2007. Proceedings 2007.

Invited Lectures/Presentations

International/National

1. *Targeting Integrin αV/β1 Receptor Manifests Intriguing Anti-Tumor Effects in Sensitive and Chemoresistant Ovarian Cancer Cells: Potential Therapeutic Target*. 64th Annual Scientific Meeting of the Society for Reproductive Investigation, Orlando, FL, March 2017.

2. *The Role of Oxidative Stress in the Pathogenesis of Ovarian Cancer*. University of Jordan, Amman, Jordan, July 2017.

3. *Novel Innovative Targets for Ovarian Cancer Therapy*. King Hussein Cancer Center, Amman, Jordan, July 2017.

4. *The Role of Oxidative Stress in the Pathogenesis of Ovarian Cancer*. King Hussein Cancer Center, Amman, Jordan, November 2016.

5. *The Role of Oxidative Stress in the Pathogenesis of Ovarian Cancer*. University of Jordan, Amman, Jordan, November 2016.

6. *New Insights for Ovarian Cancer Screening*. 4th International Conference of the Jordanian Society of Pathology and Laboratory Medicine. In collaboration with the Arabic Division of the International Academy of Pathology, Amman, Jordan, April 2011.

7. *Updates in Oxidative Stress and Ovarian Cancer*. Modern Technology Application in Pathology Conference, Amman, Jordan, July 22 – August 1, 2010.

8. *The Role of p53 in the Pathogenesis of Keloids*. International Meeting on Mechanisms Involved in Tissue Repair and Fibrosis: Role of the Microfibroblast (Differentiation and Apoptosis), Lyon, France, December 1997.

Local/Regional

1. *The Role of Oxidative Stress in the Pathogenesis of Ovarian Cancer*. Joint Annual Reproductive Sciences Retreat, Departments of Obstetrics and Gynecology, Wayne State University School of Medicine and The University of Toronto; and Annual Michigan Alliance for Reproductive Technologies and Sciences (MARTS) Meeting at Wayne State University, Detroit, MI, October 2017. Retreat

2. *Invited Guest Speaker*. Tumor Microenvironment Section, Karmanos Cancer Center, Detroit Medical Center/Wayne State University School of Medicine, Detroit, MI, June 2016.

3. *Molecular Biological Procedures*. C.S. Mott Center for Human Growth and Development, Division of Reproductive Endocrinology and Infertility Laboratory Techniques Summer Course, Wayne State University School of Medicine, Detroit, MI, September 2015.

4. *New Insights into Pathogenesis of Ovarian Cancer*. The C.S. Mott Center Summer Reproductive Sciences Technology Course, Department of Obstetrics and Gynecology, Wayne State University School of Medicine, Detroit, MI, July 2014.

5. *The Role of Oxidative Stress in the Pathogenesis of Pro-Fibrotic Gynecologic Disorders*. 4th Annual Scientific Retreat, The C.S Mott Center for Human Growth and Development, Department of Obstetrics and Gynecology, Wayne State University School of Medicine, Detroit, MI, May 2014.

6. *Release of Substrates, Cofactors, and Products of Nitric Oxide Synthase Are Altered during Oocyte Aging*. 4th Annual Scientific Retreat, The C.S Mott Center for Human Growth and Development, Department of Obstetrics and Gynecology, Wayne State University School of Medicine, Detroit, MI, May 2014.

7. *The Impact of Myeloperoxidase on Metaphase II Mouse Oocyte Quality*. 4th Annual Scientific Retreat, The C.S Mott Center for Human Growth and Development, Department of Obstetrics and Gynecology, Wayne State University School of Medicine, Detroit, MI, May 2014. **First Prize Award**

8. *Differential Expression of Glutathione Peroxidase and Glutathione Reductase in Chemoresistant Epithelial Ovarian Cancer Cells*. The Michigan Alliance for Reproductive Technologies and Science (MARTS), Fourth Annual Research Symposium, University of Michigan, Ann Arbor, MI, May 2013.

9. *The Role of Oxidative Stress in the Pathophysiology of Gynecologic Fibrotic Disorders: Postoperative Adhesions, Fibroids, Endometriosis, and Ovarian Cancer*. 1st Annual Scientific Retreat of The C.S. Mott Center for Human Growth and Development, Department of Obstetrics and Gynecology, Wayne State University School of Medicine, Southfield, MI, May 2011.

10. *New Insights in Ovarian Cancer Screening*. Department of Obstetrics and Gynecology Wayne Day: New Frontiers in the Treatment of Gynecologic Cancer, Wayne State University School of Medicine, Detroit, MI, December 2010.

11. *Molecular Characterization of Adhesion and Peritoneal Fibroblasts*. Adhesion Mini Symposium, Department of Obstetrics and Gynecology, Wayne State University School of Medicine, Detroit, MI, March 2001.

12. *Multiplex RT/PCR Technique, Concept and Application*. Center for Biomedical Research, College of Art and Sciences, Oakland University, Rochester, MI, May 1999.

13. *Techniques for Characterizing and Manipulating DNA from the Basis of Much of Modern Biomedical Research*. Department of Chemistry, Oakland University, Rochester, MI, January 1999.

14. *Bcl-2/Bax Ratio as a Measure of the Rate of Apoptosis in Keloid Fibroblasts*. Oxford Biomedical Research Inc., Oxford, MI, January 1998.

15. *PCR Techniques, Concepts and Applications*. Howard Hughes Research Program, Oakland University, Rochester, MI, May 1998.

16. *Multiplex RT/PCR Technique, Concept and Application*. Center for Biomedical Research, College of Art and Sciences, Oakland University, Rochester, MI, May 1997.

17. *Application of RT/PCR*. Department of Chemistry, Oakland University, Rochester, MI, June 1994.

Invited Seminars and Grand Rounds

1. *New Insights into the Pathogenesis of Post-Operative Adhesions Development*. Department of Obstetrics and Gynecology Grand Rounds, Georgia Regents University, Augusta, GA, January 2017.

2. *Novel Innovative Targets for Ovarian Cancer Therapy*. Cancer Center Seminar, Georgia Regents University, Augusta, GA, January 2017.

3. *The Role of Oxidative Stress in the Pathogenesis of Pro-Fibrotic Gynecologic Disorders*. Augusta Research Day, Department of Obstetrics and Gynecology Grand Rounds, Georgia Regents University, Augusta, GA, June 2013.

4. *Dichloroacetate Induces Apoptosis of Epithelial Ovarian Cancer Cells Through the Inhibition of Oxidative Stress Enzymes*. SGI-SMFM Scientific Meetings Abstract Presentations, Department of Obstetrics and Gynecology Grand Rounds, Wayne State University School of Medicine, Detroit, MI, February 2010.

5. *PCR Techniques Concepts and Clinical Applications*. Clinical Fellows Seminar, Division of Reproductive Endocrinology and Infertility, Department of Obstetrics and Gynecology, Wayne State University School of Medicine, Detroit, MI, March 2000.

6. *The Role of p53 and Apoptosis in the Pathogenesis of Keloids*. Department of Obstetrics and Gynecology Grand Rounds, Wayne State University School of Medicine, Detroit, MI, July 1998.

Exhibit B

MATERIALS CONSIDERED

Blount, A M. "Amphibole Content of Cosmetic and Pharmaceutical Talcs." *Environmental Health Perspectives* 94 (August 1991): 225–30.

"Deposition & Exhibits of John Hopkins, PhD, In Re: Talcum Power Prod. Liab. Litig., MDL No. 2738.," August 16, 2018.

"Deposition of Alice M. Blount, Ph.D., Circuit Court of the City of St. Louis State of Missouri, Case No.: 1522-CC10417-01," April 13, 2018.

"Deposition & Exhibits of Julie Pier, MDL No. 2738." In re: Talcum Power Prod. Liab. Litig., September 12, 2018.

"Expert Report of Michael Crowley, Ph.D., In Re: Talcum Powder Prod. Liab. Litig., MDL No. 2738," November 12, 2018.

Longo, William E., and Mark W. Rigler. "The Analysis of Johnson & Johnson's Historical Baby Powder & Shower to Shower Products from the 1960's to the Early 1990's for Amphibole Asbestos," November 14, 2018.

Exhibit C

Dr. Ghassan M. Saed Compensation and Prior Testimony

Dr. Saed's fees are $600/hr. He has not testified in other cases during the previous four years.